INTRACRANIAL TUMOURS.

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BY

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PREFACE.

THE following pages are based on a series of Lectures, which, in the capacity of Lecturer on Clinical Medicine in the University of Durham, I delivered, now some ten years ago, to the Students of the College of Medicine, Newcastle-on-Tyne. Portions of the work were communicated in the form of three Lectures to the gentlemen attending the Edinburgh Post-Graduate Course during the autumn of last year.

The work is essentially based upon my own clinical experience, but I have not hesitated, whenever it has seemed necessary, to avail myself of the opinions of other observers. In particular, I have to acknowledge my great indebtedness to the writings of Dr. Hughlings Jackson, of whose work on this subject British Medicine will always be justly proud.

My sincere thanks are due to Mr. A. W. Hare, M.B., F.R.C.S.E., for the chapter on the Surgical Treatment of Intracranial Tumours which he has kindly written for me; also to Drs. Clouston and Carlyle Johnstone for the remarkable specimens of Intracranial Aneurism which are represented on pages 165 and 168; to Dr. T. W. McDowall for the Brain represented in Fig. 113; and to Dr. David Drummond for the cuts of his cases of Perforating Sarcoma.

23 DRUMSHEUGH GARDENS,
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CHAPTER I.

DEFINITION — INTRODUCTION — ETIOLOGY — GENERAL PATHOLOGY AND PATHOLOGICAL PHYSIOLOGY.

Definition.—Under the term intracranial tumour, it is customary to include all new growths which originate within, or are contained in, the cavity of the cranium.

Localised inflammatory thickenings or deposits, more especially when syphilitic and tubercular, are, in some cases, with difficulty distinguished from the more sharply defined new growths—tumours properly so called; indeed the distinction may be impossible, for the two conditions are often combined, and may insensibly run one into the other.

Cerebral abscesses are not included under the definition; for, although in some cases it may be difficult or impossible to differentiate a chronic abscess from a cerebral tumour during life, the distinction is readily enough made post mortem.

Large aneurisms and parasitic cysts must be regarded as intracranial tumours.

Simple cysts, as well as cysts which develop in connection with gliomata and other forms of new growth, are usually described in connection with intracranial tumours.

Introduction.—The subject of intracranial tumours is of great interest, from a clinical, physiological, and pathological point of view.

The brain and its membranes are favourite situations for the formation of new growths. In no other situation in the body is such a great variety of tumours met with; it would almost indeed seem that the delicate and soft brain tissue, richly supplied as it is with blood-vessels and lymphatics, is as fine a forcing and feeding ground for new growths as Koch's nutrient jelly is for micrococci and allied organisms. Syphilitic and tubercular tumours, gliomata, and the various forms of sarcoma, are most frequently met with; different varieties of cancer, endotheliomata (cylindromata), and psammomata are not uncommon; while cholesteatomata, parasitic cysts (*Echinococci* and *Cysticercus Cellulosæ*), melanomata, osteomata, fibromata, lipomata, and neuromata are more rare.

New growths may occur in any part of the intracranial cavity. In some cases the tumour is single, in others multiple. The tumour may derange the functions of the great intracranial nerve centres as a whole; or it may invade, and irritate or destroy, individual parts—limited portions of grey matter, conducting tracts, or peripheral (cranial) nerves. The symptoms are in some cases of the most diverse and complicated character; and the diagnosis may be attended with great difficulty. But in no disease is an exact diagnosis of more importance; for, while experience has abundantly shown that it is impossible to cure intracranial tumours (the syphilitic variety in many cases excepted) by any internal remedies with which we are at present acquainted, the results obtained by Hughes Bennett, Godlee, and Victor Horsley go far to prove that in some cases at all events intracranial tumours may be satisfactorily removed by operation. But since the removal of the new growth is in many instances altogether impossible, a correct diagnosis is obviously essential for successful interference. The exact localisation of the tumour is necessarily a point of the first importance; while a knowledge of the pathological character of the new growth may enable us to decide whether an operation is feasible or not.

Etiology.—The causation of intracranial tumours differs in no way, so far as I know, from the causation of tumours in other parts of the body.

In some cases the new growth is primary, in others secondary; in

the latter, the primary tumour may be situated in any part of the body, but secondary deposits of new growth in the intracranial nerve centres are, I think, especially apt to follow similar diseased conditions of the pulmonary tissue. This is probably due to the fact that when the primary new growth is situated elsewhere, say in the abdomen, the liver and the lung act as filters, and protect the brain, catching as it were any minute particles of malignant growth which get into the venous circulation; whereas, when the lungs are themselves the seat of the disease, any infective particles which get into the circulation, are carried to the left heart, and may consequently find their way, through the arterial system, to the brain as well as to the other organs. This explanation of course applies only to those cases in which the secondary deposits are carried to the brain through the blood-vessels.

It is unnecessary to say anything with regard to the hereditary and general constitutional causes which predispose to tumour formation.

Amongst the more direct causes, injury occupies an important place. There can, I think, be little doubt that blows and falls on the head do sometimes lead to the formation of gliomatous, and perhaps also of sarcomatous tumours; syphilitic tumours, too, not infrequently appear to owe their exciting cause to a head injury; and in two instances I have known a scrofulous tumour to follow a severe blow upon the head, the new growth developing either immediately below the seat of the injury, or at the point of contre-coup. The explanation of such cases is probably this, that the blow produces a local inflammatory lesion or contusion, which forms a suitable nidus for the development of the tubercular germs (tubercle bacillus or its spores) which are already circulating through the system.

How far mental anxiety and worry, or other conditions which depress and exhaust the cerebral nerve centres, and how far alcoholic excesses and other conditions which are attended with repeated cerebral congestions, may predispose to, or aid in, the formation of brain tumours, is altogether uncertain. Whether, too, any of the new growths which occur within the cavity of the cranium owe their origin to micro-organisms is unknown; but I have for long thought it probable that the cause of some forms of glioma is of this nature.

Intracranial tumours are more frequent in the male than in the female; in some cases this is doubtless due to the fact that men are more frequently affected with syphilis, and are more exposed to head injuries, and possibly because they are more addicted to alcoholic excesses than women.

For the same reason, those persons whose occupation exposes them to head injuries, and possibly to mental worry and anxiety, are perhaps somewhat more liable to be affected with intracranial tumours than other members of the community. Occupation is, however, a matter of very little consequence from an etiological point of view.

Age is of more importance, for undoubtedly some forms of tumour are more likely to appear at one age than at another. Scrofulous tumours are more common in the young; syphilitic tumours in adults, the period of greatest frequency being probably between the ages of thirty and forty-five; cancers of the brain, like cancers elsewhere, are more common in old people; gliomatous tumours may occur at any age, but in my experience are more frequently seen in young persons or in adults—*i.e.*, not in *old* people.

The general pathology and pathological physiology of brain tumours.—The number of symptoms which may be produced by intracranial tumours is very great, and the manner in which they may be grouped and arranged most diverse. Hence the clinical picture which individual cases present may be very different. Now, since the exact nature of the symptoms which are present in any given individual case depends upon the effects which the new growth has upon (*a*) the intracranial contents, and (*b*) the cranial walls, it follows that the intelligent comprehension of this difficult part of the subject will be greatly facilitated if we start with clear notions as to the effects which intracranial tumours in general may produce upon the brain and other structures contained within the cavity of the cranium.

In the *first* place, it is essential to remember that, after the sutures have been ossified, the cavity of the cranium is, for all practical purposes, a closed cavity; and that, when a tumour or new growth is developed in any part of it, the intracranial pressure must necessarily be increased, and the brain, and other structures contained within it, must necessarily be compressed, unless compensation is

effected, and increased pressure and squeezing prevented, either by atrophy of intracranial structures, displacement of intracranial fluids, or yielding of cranial walls, *i.e.*, separation of the sutures.

Further, it must be remembered that the general cavity of the cranium is divided by the tentorium cerebelli into two separate parts; and that while tumours growing above the tentorium chiefly compress the parts which are situated in the upper cranial cavity, and as a rule exert comparatively little pressure upon the cerebellum, pons Varolii, and medulla oblongata (*i.e.*, the parts in the lower cavity), the reverse is by no means the case. Subtentorial tumours, in fact, may not only exert very great compression on the parts in their immediate neighbourhood (cerebellum, pons Varolii, and medulla oblongata), but may, and very frequently do, by producing dropsy of the ventricles, cause an enormous increase of pressure in the upper cavity, or, as we may term it, the cavity of the brain proper. Nay, further, a small tumour or cicatricial inflammatory product, which produces comparatively little direct (pressure) effects upon the parts below the tentorium (*i.e.*, in its immediate neighbourhood), may cause great distention of the ventricles and enormous increase of the general intracranial pressure.

In a less perfect manner, the upper cranial cavity is divided by the falx into two separate cavities; and by means of the resistance which this tough, partitioning membrane affords, the cerebral hemisphere on one side is to some extent protected from the pressure effects of a large, rapidly increasing tumour on the other.

As a matter of fact, flattening of the convolutions, obliteration of the sulci, anæmia of the surface of the brain, a dry, sticky condition of the membranes, and in many instances dropsical distention of the ventricles with œdema of the brain tissue—in other words, the post mortem evidences of increased intracranial pressure—are met with in the great majority of cases of intracranial tumour after death; and it is, I believe, to this increased intracranial pressure and its results that the *general* symptoms of intracranial tumour, such as headache, vomiting, double optic neuritis, vertigo, mental dulness, lethargy, &c., are largely due.

The exact causation of the double optic neuritis is a matter of dispute. Personally I am of opinion that increased intracranial

pressure is in many cases an important, though not the sole or perhaps the most important, factor in its production. This point will afterwards be considered more in detail.

The term *general* is here used to indicate those symptoms which result from the presence of a tumour in any part of the cranial cavity, as distinct from the *localising* symptoms, which are due to the effects which the tumour produces on special parts, and which are indicative of its exact locality or seat.

As we shall afterwards see, headache, when localised—and more especially when associated with tenderness on skull percussion—vomiting and vertigo, when obstinate and severe, may have a localising value; but in most instances these symptoms are general rather than localising symptoms, and cannot be taken as guides to the position of the new growth.

Now the degree of the increase of the intracranial pressure and the extent of the compression to which the intracranial contents are subjected, depend upon (*a*) the size, and (*b*) the rapidity of the growth of the tumour; together with (*c*) the presence or absence of dropsical distention of the ventricles and the extent thereof.

In the case of tumours which grow rapidly and are of large size—which, in other words, are attended with great increase of the intracranial pressure—we should expect the general symptoms of tumour (headache, vomiting, double optic neuritis, &c.) to be well marked; and such in fact is usually the case. Towards the later periods of cases of this description, and in many cases in which there is dropsy of the ventricles, the headache and other symptoms, which in the earlier stages may have been well-marked and characteristic, often become less prominent, or may altogether disappear, in consequence probably of the fact that, owing to the anæmia, œdema, and compression of the nervous tissue, the sensibility of the brain is diminished, the feelings and perceptions are dulled, and a condition of hebetude, mental apathy, semi-coma, or actual coma, is produced.

In those cases, on the contrary, in which the tumour is of small size or of slow growth; in cases in which, as the tumour grows, the brain tissue atrophies; in cases in which the fontanelles are not yet closed, and yielding of the bones of the skull readily takes place; in

other words, in those cases in which there is little or no increase of the intracranial pressure, the general symptoms of tumour may be altogether absent or little marked.

Another factor which probably plays an important part in the production of the "general" symptoms is the "*irritation*" which the tumour produces in the nervous tissues and in the cerebral membranes.

In some cases of intracranial tumour, as I shall afterwards point out, a diffused irritative change—which causes enlargement of the nervous tissues surrounding the tumour, and is chiefly characterised on microscopical examination by increased size of the connective tissue corpuscles or Deiter's cells—is found throughout the brain tissue; but more especially in the hemisphere or part of the brain in which the tumour is situated.

In some cases the membranes seem to be involved in this process of irritation, and there is distinct naked-eye evidence of thickening and chronic meningitis. In others, in which no such naked-eye appearances are to be seen, it is said that inflammatory changes in the meninges can be demonstrated by means of the microscope. I cannot say that I have been able to satisfy myself with regard to this point. In many of the cases of intracranial tumour which have come under my notice there have been no distinctive naked-eye appearances of diffused meningitis; while in the great majority the usual post-mortem signs of increased intracranial pressure have been prominent. In the cases of this kind which I have subjected to careful microscopical examination, I have failed to find distinctive evidence of meningitis; but I have in most cases detected enlargement of the connective tissue corpuscles, and evidences of irritative changes in the brain tissue surrounding the tumour. In some cases, I have satisfied myself that these irritative changes were wide-spread.

What share this diffuse irritative change in the neighbourhood of the tumour may take in the production of the general symptoms (headache, vomiting, double optic neuritis, &c.), I am not prepared to say. That it has some influence is probable; but, so far as I can judge, it is not such an important factor as meningeal irritation and increased intracranial pressure.

In the *second* place, the new growth necessarily presses upon or involves special structures and parts; and it is to the derangements which are produced in the structures, which are thus pressed upon or involved, that another great and most important group of symptoms —the *localising* symptoms, as they are termed—is due.

The exact nature of the localising symptoms depends upon the following points:—

(1.) The function of the particular part of the cerebrum, cerebellum, pons, medulla, intracranial nerves, blood-vessels, membranes, bones, &c., which is pressed upon or involved.

This point is self-evident, and needs no amplification. It is quite obvious that in a case in which the tumour is situated in the neighbourhood of the fissure of Rolando, and in which the motor centres are pressed upon or involved, the symptoms will be quite different from a case in which the tumour involves the præfrontal or occipital lobes; and that the exact character of the paralyses or spasms, which are present in the former case, will depend upon the exact portion of the motor area which happens to be affected.

(2.) The manner in which the part which is pressed upon or involved by the tumour is affected—whether irritated or destroyed—and, more especially in the case of destroying lesions, the extent of the changes which are induced in it.

Speaking generally, it may be stated that the effect of all lesions on the nervous tissues is to produce one or other of two results, viz., either diminution or abolition of function on the one hand, or increase of function on the other. It must, however, be remembered that the increased function, which results from pathological causes, is seldom a simple increase; as a rule, the function is not only increased, but is also perverted in character. Paralysis, anæsthesia, and dementia are good examples of diminished or abolished function; convulsions, neuralgia, and mania of increased and perverted function.

In some cases in which the symptoms are indicative of increased function, the primary lesion is probably, as Dr. Hughlings Jackson has suggested, a "destroying" one, *i.e.*, a lesion which produces diminution or destruction of function. It would appear, for example, that in some cases of mania the highest and most specialised parts of the cerebral nerve apparatus are put *hors de combat*, with the result

that the lower cerebral centres are not restrained as they are in health, the net result being symptoms of a maniacal character. In cases of this description, Dr. Hughlings Jackson supposes that the *increased* function is apparent only, and that the increase is *in reality* due to the fact that the function of the highest (controlling) centres is *destroyed, diminished, or temporarily suspended* by the lesion.

Now, whether a tumour which is pressing upon or involving a portion of brain tissue will produce diminution or exaltation of function, would appear to be due to (*a*) the rapidity with which it is growing; (*b*) its pathological nature; and (*c*) the composition, so to speak, of the nerve tissue with which it is in contact, *i.e.*, whether grey or white matter. Whether the pressure is continuous or intermittent is doubtless also a factor of importance. Other things being equal, a rapidly growing tumour is more likely to produce irritation of nerve tissue, and particularly of grey nerve tissue, than a slow growing one; while a tumour of slow development is much more likely to produce a simple atrophy of the tissue on which it presses.

Further, as we shall afterwards see, irritation of grey matter of necessity gives rise to symptoms—granting of course that the irritation is sufficiently great to excite discharge, and that the way out for the nerve force is not obstructed—while destruction of grey matter is frequently compensated and unattended by any external manifestations. Again, as we have already seen, a rapidly growing tumour is more likely to produce increased intracranial pressure and diffuse irritative changes than a slow growing one. For all these reasons, then, a tumour of rapid development is more frequently attended by symptoms both general and localising than a tumour of slow growth.

The pathological nature of the tumour is also of great importance in determining whether irritation or destruction will result in the nerve tissue which is implicated.

Those tumours which are surrounded by a capsule, which do not infiltrate the tissues with which they come in contact, and which have little or no tendency to produce meningitis (such as fibrous, fatty, and cystic tumours, some sarcomata, &c.), are much less likely to produce irritation than those new growths (such as gliomatous, syphilitic, tubercular, cancerous tumours, and some varieties of sarcoma) which produce the opposite effects—*i.e.*, which do infiltrate

the structures with which they come in contact, or which have a strong tendency to produce cerebritis or meningitis.

Encapsuled tumours are as a rule of slow growth, but infiltrating tumours, though they usually develop rapidly, do not always do so. I have, with Dr. Leslie, reported a case of infiltrating glioma in which the duration was certainly five years; and another case of the same kind, in which the duration was perhaps even longer.[1]

But whether symptoms will result from the irritative or destructive changes which a tumour produces in the nerve tissue on which it presses, or through which it infiltrates, depends to a large extent upon the fact whether that nerve tissue is composed of grey or white matter. For it is important to remember, on the one hand, that discharge of nervous energy is much more readily produced by irritation of nerve cells than of nerve tubes; tumours which are situated on the surface of the brain (such as tubercular and syphilitic deposits), and which involve and irritate the grey matter, are therefore much more likely to be attended with "irritative symptoms" (spasms, flashes of light, &c.), than tumours which involve and irritate nerve tubes, say in the centrum ovale, internal capsule, or peripheral nerves.

On the other hand, destruction of grey matter is much more easily compensated than destruction of conducting fibres or nerve tubes. Destruction of a part of the motor cortex, indeed of a very large part, as the specimen represented in Figs. 1, 2, 3, and 4 shows, may be quite unattended with symptoms (*i.e.*, paralysis), but a limited destruction of the conducting fibres, say of the internal capsule, is, as far as we know, almost certain to be followed by paralysis.

Whether permanent paralysis will result from destruction of motor-conducting fibres is also largely dependent upon the degree of specialisation, so to speak, of the movements with which those fibres are concerned. Destruction of the fibres which innervate the muscles of the thumb and fingers will produce more lasting and permanent paralysis than destruction of the fibres which supply the muscles of the upper arm and shoulder. For the same reason, as will be afterwards more fully explained, compensation is more readily effected in the lower than in the upper extremity, for the movements of the leg are

[1] *Edinburgh Medical Journal*, January 1887, pp. 591 and 623.

more automatic (less highly specialised and less independent of those of the opposite leg) than the movements of the arms.

The effects of "destroying" and "discharging" lesions, and the important subject of "compensation," will be afterwards considered more in detail.

In considering the local alterations which the tumour produces in

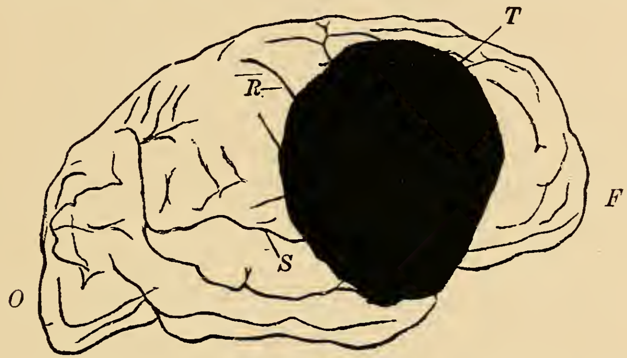

FIG. 1.—Diagrammatic outline of the right hemisphere of the brain in the case of M. D., showing the exact position of the lesion (the dark area to which the letter *T* points).

The letter *F* points to the frontal and *O* to the occipital end of the brain; *R* to the fissure of Rolando, and *S* to the fissure of Sylvius; *T* to the tumour.

The patient, a girl, aged twenty, was admitted to the Newcastle-on-Tyne Infirmary under my care on February 5th, 1877, suffering from headache, vomiting, and giddiness. There was well-marked double optic neuritis, but absolutely no paralysis. Under iodide of potassium great improvement took place, and the patient felt so well that she had made arrangements to leave the hospital. The very night before she was to go home, she was seized with an epileptiform convulsion, in which she died.

At the post-mortem examination the very large tumour, represented in Figs. 2, 3, and 4, was found; it had produced very extensive destruction of the motor centres, and yet there was never the slightest trace of paralysis.

The case is reported in full in the *Edinburgh Medical Journal* for February 1879, p. 693.

the parts with which it is in direct contact, it is necessary to remember that the symptoms which result are not always *directly* due to the derangement of function in the part which is immediately implicated, but that they may be the result of *indirect and secondary* alterations in some other and distant part of the nervous tissue.

The point is one of practical importance, for symptoms produced in this way may be localised and limited in extent, and may give an erroneous idea of the position of the tumour.

Symptoms of this kind—*pseudo-localising* symptoms, as they may

perhaps be termed—are, I believe, chiefly produced in one or other of the following ways.

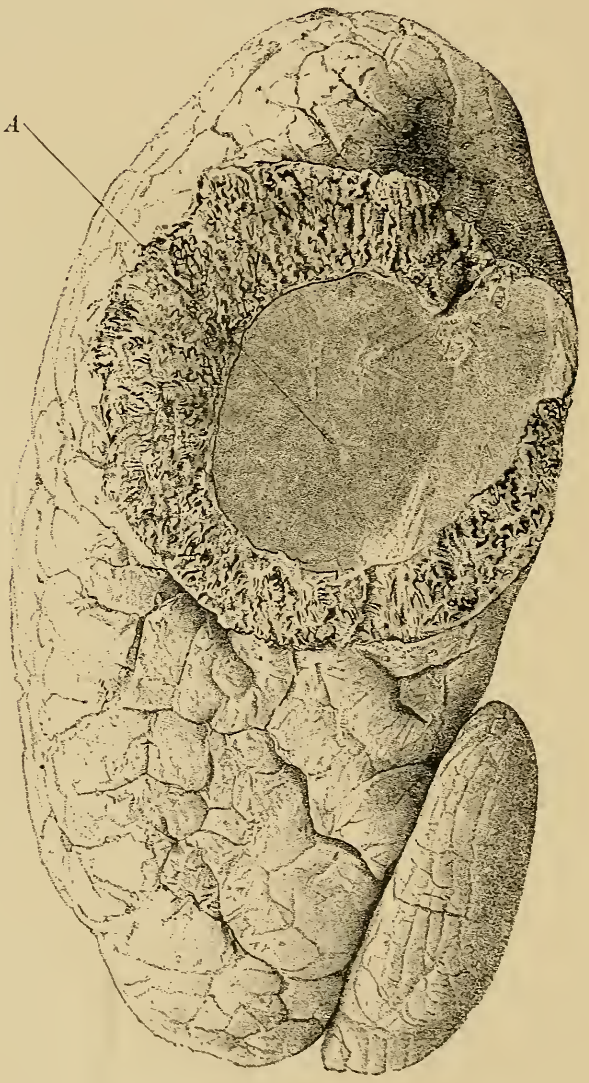

Fig. 2.—The outer surface of the right hemisphere of the brain in the case of M. D. (large sarcoma growing from the dura mater, which had produced extensive atrophy of the motor centres, but was unattended with paralysis), showing the tumour *in situ*. (Copied from a photograph and somewhat reduced in size.)

The letter *A* points to the outer surface of the dura mater over the centre of the tumour.

(1.) *By alterations of the blood vascular supply to distant centres and parts.*—A syphilitic gumma at the base of the brain, for example,

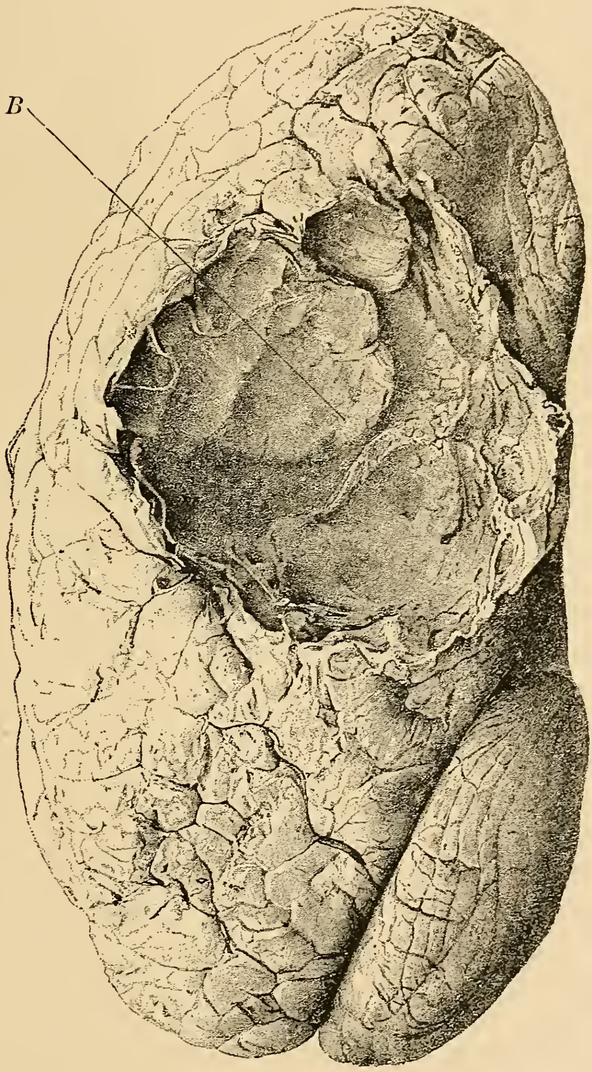

Fig. 3.—The outer surface of the right hemisphere of the brain in the case of M. D., showing the brain after the tumour was removed. (Copied from a photograph, and somewhat reduced in size.)

The letter *B* points to the extensive depression on the surface of the brain, into which the tumour fitted.

which involves and destroys the third nerve, may at the same time surround and obstruct the middle cerebral artery, and produce ex-

Fig. 4.—Transverse vertical section through the right hemisphere of the brain in the case of M. D., showing the tumour in section, and the depression in the brain tissue produced by it. (Copied from a photograph, and somewhat reduced in size.)

The letter *A* points to the tumour ; *B* is placed on the inner (left) side of the right hemisphere, which has been separated from the opposite (left) hemisphere by a section made from before, backwards in the middle line.

tensive softening of the motor convolutions ; the resulting paralysis—hemiplegia on the opposite, and paralysis of the muscles supplied by the third nerve on the same side—is suggestive of a chronic lesion of the crus cerebri. Other illustrative examples might be given, but this point is so obvious that further details are unnecessary. Possibly the vascular supply of a distant part of the brain tissue may be altered, and the nutrition and function of that centre affected, by the pressure of a tumour upon the vaso-motor nerves distributed to the blood-vessels.

(2.) *By indirect (reflex) irritation or inhibition of distant centres.*—In cases of this description the tumour produces alterations, structural and functional, in the centre with which it is immediately

in contact (centre *A*), with the result that a distant centre (*B*) is either irritated or inhibited. The symptoms which are produced in this manner show that the function of the distant centre (*B*) is deranged; hence the observer may conclude that the tumour is situated in the immediate neighbourhood of that centre.

That the function of a part of the brain which is itself free from disease may be deranged (excited or inhibited) in consequence of disease in some other and distant part, seems certain. It is probable that in some cases of mental derangement the symptoms are produced in this way; and the phenomena of certain cases of aphasia seem to show that lesions on the sensory side of the speech mechanism (sensory speech centres) almost of necessity produce derangements in the motor (speech) centres. Possibly, too, the nervous derangements (paralyses, spasms, &c.) which are met with in some cases of hysteria may be best explained in this manner; but that such coarse phenomena as localised spasms and localised paralyses are frequently or usually produced in this way, as Brown-Sequard in particular would have us suppose, seems to me altogether improbable. The risk of erroneous diagnosis (localisation) from this cause has, in my opinion, been exaggerated. When, in short, in a case of intracranial tumour the symptoms clearly point to derangement of function in a localised and limited portion of brain tissue, the physician will, I think, be wise to give the fact its full localising value. In the majority of cases of this description the symptoms are directly due to deranged function in the nerve tissue which is in the immediate neighbourhood of the tumour, or of some nodule of new growth in the case of a multiple lesion. Irritation is chiefly produced in the nervous tissue which surrounds the tumour. The irritative phenomena, therefore, do not so directly point to the exact position of the new growth as the symptoms due to destruction. In those cases in which no such direct relationship exists, a sufficiently carefully conducted post-mortem examination (naked eye and microscopic) may be expected to show distinct pathological changes in the part whose function was deranged, and, in some instances at all events, to demonstrate the manner in which this apparently independent lesion had indirectly resulted from the presence of the new growth —*i.e.*, to show, for example, that the tumour had involved the vessels

supplying the affected part, or that it had so altered the relationship of the parts within the cranium as to produce localised pressure on the affected part, on the vessels supplying it, or on the conducting tracts proceeding from it, &c.

It is quite common, in fact usual, to find the hemisphere in which the tumour is situated larger than the opposite one. In some cases this increase in size is accounted for by the size of the tumour itself; in others it is due to œdema or inflammatory swelling around the

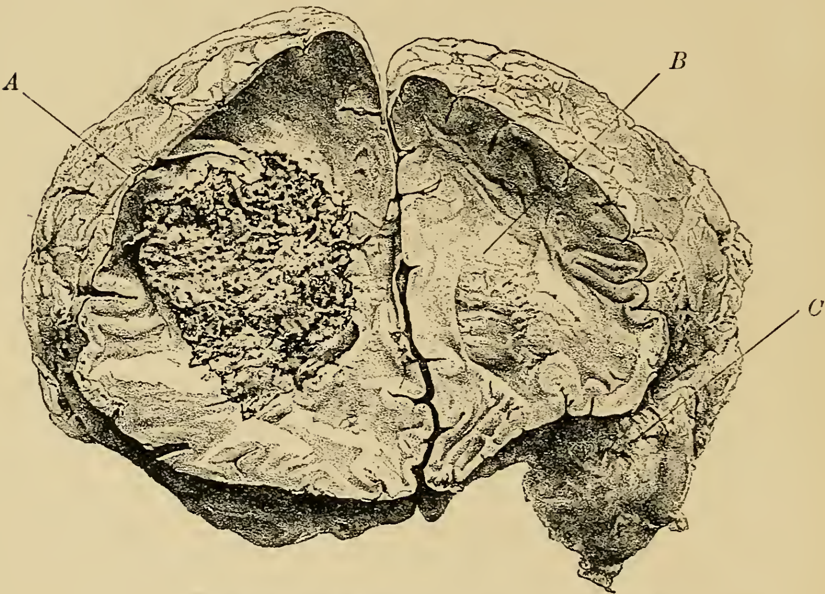

Fig. 5.—Transverse vertical section through the frontal lobes in a case of tumour (sarcoma) of the right hemisphere.

The right frontal lobe in which the new growth (to which the letter *A* points) is situated is markedly larger than the left.

The letter *B* points to that part of the left hemisphere which is situated just in front of the lateral ventricle; *C*, to the tip of the left temporo-sphenoidal lobe.

new growth; but in many cases it seems to be due to the chronic diffuse (irritative) change which has been previously referred to.

In consequence of the swelling which is produced in one or other of these ways, the relationship of the different parts contained within the cranium may be considerably altered; and, as a result of stretching, twisting, or squeezing, the functions of portions of the nerve tissue, at a distance from the tumour, may be deranged, or their vascular supply interfered with.

In Figs. 5 and 6, the swollen condition of the hemisphere in which the tumour is situated is well shown.

FIG. 6.—Transverse vertical section through the brain, showing a large glio-sarcomatous tumour in the left hemisphere, which is markedly larger than the right.

The letter *A* points to the nucleus caudatus; *B*, to the optic thalamus; and *C*, to the lenticular nucleus of the right side.

The letter *D* points to the left or internal capsule, which was compressed, and to some extent invaded by the new growth; the letter *E* points to the tumour.

(3.) Cerebral symptoms in cases of intracranial tumour may be due to associated meningitis, hæmorrhage into the substance of the tumour and surrounding nerve tissue, or other accidental complications.

(4.) The presence or absence of constitutional disturbance, and the nature of the general constitutional symptoms, depend partly upon the character, duration, and severity of the nervous symptoms (pain, sleeplessness, vomiting, dysphagia, &c.); partly upon the pathological nature of the new growth; but are chiefly due, more especially in the earlier stages of the case, to associated lesions and to the condition of the abdominal and thoracic viscera.

(5.) The severity, both of the nervous and constitutional symptoms, depends to some extent upon the individuality, so to speak, of the patient. The capability which different individuals possess of bearing pain and of resisting disease is, as we all know, very different. The reaction, both to physiological and pathological stimuli, is very different in different cases. It is reasonable, therefore, to suppose that a cerebral lesion (tumour), which in its early stages produces in one individual comparatively trifling symptoms, may give rise in another to profound disturbances (pain, sleeplessness, emotional or mental derangement, &c.).

CHAPTER II.

SYMPTOMATOLOGY AND CLINICAL HISTORY—ANALYSIS OF INDIVIDUAL SYMPTOMS.

Clinical Pictures presented by different Groups of Intracranial Tumours.

Although the symptoms which are met with in different cases of intracranial tumour are very different, and the manner in which those symptoms are arranged in individual cases is subject to great variation, it is, I think, possible to place most cases in one or other of the four following groups.

1.—*Cases of intracranial tumour in which the presence of the tumour is not indicated by any characteristic symptoms during life.*

Intracranial tumours are sometimes found post mortem whose presence was not suspected during life. In the great majority of the cases of this description there are cerebral symptoms.

In some, these symptoms are characteristic; and the failure in diagnosis is due, either to the circumstance that the facts on which the physician has to base his opinion are wanting (as where a patient is brought into hospital in a state of coma, and dies before a satisfactory history of the case is obtained), or to erroneous observation or deduction on the part of the medical attendant.

In other cases, the symptoms produced by the tumour are masked by those of some associated lesion or complication—such, for instance, as cerebral meningitis or cerebral hæmorrhage.

In very rare and quite exceptional cases, cerebral symptoms are absolutely wanting. One of the most remarkable cases of this kind, with which I am acquainted, was that of my old teacher, the late Professor John Hughes Bennett, who died after the operation of lithotomy. He was, as is well known, one of the greatest clinical teachers of his time; and his keen intellectual powers were, I understand, unimpaired even to the end. He certainly never manifested any

symptoms suggestive of an intracranial tumour; nor did any of the many eminent physicians whom he consulted for his laryngeal and other troubles ever suspect the presence of such a lesion; and yet at the autopsy, a tumour the size of a hen's egg was found two inches above the right ear. It was situated between the dura and the bone, and projected towards the brain so as to produce a deep pit or hollow, into which it fitted. The parietal bone over the tumour was thinned by pressure and absorption; at one point, about the size of a shilling, all trace of bone had disappeared, the osseous tissue being replaced by fibrous membrane; around the circumference of the tumour the bone was thickened and hypertrophied. The dura mater over the tumour was thickened. The convolutions were flattened and pressed down, but not otherwise altered; there was no congestion and no softening in the neighbourhood of the new growth.[1] The tumour was evidently stationary, or of very slow growth. Dr. A. Hughes Bennett tells me that the tumour was probably congenital, for the Professor had frequently directed his attention to a depression of the skull over the position of the tumour, and had stated that it had existed since childhood.

We must conclude, then, that in some rare cases (in which an intracranial tumour is stationary or of extremely slow growth; in which there is no increase of intracranial tension; and no irritation in the neighbourhood of the lesion) all symptoms may be entirely wanting. I repeat, however, that in the great majority of cases, in which the tumour appears to be completely "latent" (*i.e.*, unattended with any symptoms), there are some symptoms, though they are not in all cases suggestive or characteristic of the presence of a new growth. In short, I agree with Obernier in thinking that "every tumour" (I should say every *growing* tumour) "of any size, which presses upon or injures the brain tissue, must give rise to symptoms, either transitory or otherwise."[2]

In speaking of the absence of symptoms in some cases of intracranial tumour, Dr. Hughlings Jackson makes the following very suggestive remarks:—"Yet, when there is no striking defect, there may be dulling of mind (slowness, hebetude, incapacity for continued mental exertion); still there are no symptoms which make the

[1] *British Medical Journal*, 9th October 1875, p. 453.
[2] Ziemssen's *Cyclopædia of Medicine*, vol. xii., p. 241.

patient seek a doctor until perhaps shortly before death. The patient's mind may be so generally reduced to a lower level that neither he nor his friends notice any failure; or they may notice 'alterations' of disposition, which they put down to wrong causes—to the influence of external causes. There is doubtless, from every degree of lesion of the cerebral hemisphere, loss of some special or 'delicate' intellectual process, and correspondingly of some of the finer emotions—for every intellectual act is accompanied by emotion. To adopt an expression from ophthalmology, there is no doubt a 'limitation of the mental field,'—a limitation of the idea of consciousness." [1]

2.—*Cases in which the characteristic symptoms, indicative of the presence of a tumour in some part of the intracranial cavity, are present, but in which there are no symptoms indicative of its exact site.*

In typical cases of this kind, headache, vomiting, and characteristic changes in the optic discs (double optic neuritis, or optic atrophy) are present; in many cases there is also vertigo.

In the majority, a careful comparison of the previous with the present mental state will show some loss of mental power, some change of disposition, some derangement of intellect or mental power, some disturbance of the emotional faculties, or some loss in the power of self-control. In not a few cases the mental alteration is profound. In most cases of this kind the mental symptoms are of a negative kind; the intellectual deterioration may vary from a slight loss of memory, or mere dulling, as it were, of mental acuteness on the one hand, to complete loss of mental power (dementia or coma) on the other. In other cases there is irritability of temper; and in a few—but these are exceptional and rare—delusions, excitement, or even violent outbursts of acute mania occur. A more detailed description of the different forms of mental alteration which may occur in this and the two following—third and fourth clinical groups of intracranial tumours—is reserved until the individual symptoms are analysed.

In many cases an excess of phosphates is present in the urine. In some the appetite is voracious. Pseudo-apoplectic attacks, or true

[1] Lectures on the Diagnosis of Tumours of the Brain, *Medical Times and Gazette*, 23d August 1873.

apoplectic seizures—the result of extravasations of blood from the thin walled vessels, which are abundantly present in some forms of tumour—may occur.

General epileptiform convulsions are sometimes observed; and in some cases the patient dies during an attack of this kind.

3.—*Cases in which the symptoms show not only that a tumour is present in some part of the intracranial cavity, but in which they indicate, more or less closely, its exact locality or site.*

In this group of cases, in addition to headache, vomiting, double optic neuritis, or post-neuritic optic atrophy, &c. (see the symptoms described under group 2), symptoms indicative of functional disturbance *of special parts of the intracranial contents* are also present. These symptoms may be due to functional derangement (1) of limited portions of grey matter; (2) of the great conducting paths as they pass through the intracranial nerve centres; (3) of involvement of one or more of the cerebral nerves within the cavity of the cranium; or (4) implication of localised portions of the cranial walls.

These "*localising*" symptoms, as they are termed, are numerous. The more important are—localised paralyses or spasms of the muscles of the limbs, face, or trunk; localised derangements of the tactile sensibility of the skin (anæsthesia or hyperæsthesia), of the limbs, face, head, or trunk; localised headache, more especially when, in addition to the headache, there is localised tenderness on percussion of the painful part; derangements of the sense of sight, other than the dimness of vision and limitation of the visual field due to peripheral causes — in other words, the different forms of hemianopsia, amblyopia, or amaurosis, not due to recognisable ophthalmoscopic changes in the optic discs; derangements of the sense of hearing, smell, or taste, other than those which result from peripheral causes; the various forms of aphasia; special forms of mental deterioration, such, for instance, as the loss of the power of mental concentration, which seems to result from a lesion of the frontal lobes; urgent and obstinate vomiting; marked vertigo (when due to central causes); a reeling (cerebellar) gait; polyuria and glycosuria (the result of central causes); enlargement of the head; marked difficulty in swallowing, &c.

The bilateral dimness of vision (both loss of acuity of vision and contraction of the fields), which is the result of optic neuritis or post-neuritic optic atrophy, has no localising value; it is one of the common symptoms of the second clinical group.

Localised paralyses in the muscles of the trunk are rarely observed as the result of chronic cerebral lesions, for, as Dr. Broadbent has shown, muscles which act in concert can be put into action from either hemisphere. Localised spasms in the trunk muscles do probably occur in some cases, but often pass unnoticed.

By means, then, of these localising symptoms—and more particularly by observing the manner in which they are grouped together—it is possible in some cases to form a very accurate conclusion as to the exact locality in which the tumour is situated. In some cases (as, for example, when the "general" symptoms of a tumour are present, together with hemiplegia) it may not be possible to say more than that the tumour involves this or that cerebral hemisphere. In others, it is possible to locate the tumour in the cerebellum, pons Varolii, or medulla oblongata; in others, to say that it is situated at the base, or in the frontal, parietal, temporo-sphenoidal, or occipital lobe; in others, that it involves the corpus callosum; and in some, that it implicates special and limited areas (centres) of the motor cortex.

The more detailed cónsideration of the special symptoms indicative of lesions of these different parts is reserved until the diagnosis is discussed.

4.—*Cases in which there are distinct indications of derangement or disease of the intracranial contents, and in which the symptoms may be due to the presence of an intracranial tumour, but are not typical and characteristic of that condition.*

The cases included in this group often give rise to great difficulties in diagnosis.

In many, the symptoms, taken as a whole, are strongly suggestive of the presence of an intracranial tumour, but some striking and characteristic symptom, which is necessary to clinch the diagnosis, is wanting; hence there is difficulty and doubt.

The absence of characteristic changes in the optic discs is the fact which, above all others, is apt to give rise to difficulties of this kind.

The absence of double optic neuritis or post-neuritic atrophy does not necessarily exclude tumour, though it certainly throws doubt upon the diagnosis.

If, in addition to the fact that there are no ophthalmoscopic appearances, localising symptoms are also wanting, the doubt is still further increased.

But further, when the headache, vomiting, and other "general" symptoms are not urgent and prominent, the doubt may become so great, that it may be impossible, without jumping at conclusions and mere guessing, to be dogmatic, and give a positive opinion as to the nature of the case. The difficulty is of course intensified when the patient is naturally of a nervous, emotional, or hysterical temperament; and in those cases in which hypermetropia, anæmia, ovarian irritation, or some other condition, which is a possible cause of headache and vomiting, is also present.

In other cases, some symptom (such as localised paralysis or spasm) clearly indicative of a localised brain lesion, which may be a tumour, is present, but the general symptoms of tumour are either altogether absent or only slightly marked. Optic neuritis may be wanting; or headache, vomiting &c., may be altogether absent or very slight.

In others, again, the mental disorganisation or derangement may be so great that the symptoms of tumour, even if they are present, are thrown into the background or are not complained of. In cases, for example, in which profound dementia, or the much rarer symptom acute mania, is associated with an intracranial tumour, the physician who has not had the opportunity of watching the case from the commencement, or who has no satisfactory record of the previous history and condition, may never suspect the presence of a new growth.

For the same reasons, the presence of well-marked symptoms of hysteria, meningitis, or cerebral hæmorrhage, or the fact that the patient is markedly hypermetropic, profoundly anæmic, or suffering from Bright's disease or plumbism, may throw the physician off his guard, and lead him in some cases, very naturally (and perhaps not illogically), to overlook the presence of the tumour, and to conclude that the symptoms are entirely due to the presence of the associated disease or pathological condition.

These difficulties will be still further appreciated when the differential diagnosis of intracranial tumours is more minutely considered.

Analysis of Individual Symptoms.

After this general sketch of the more important symptoms which may be present in the different clinical groups into which I have divided intracranial tumours, it will be well to examine the individual symptoms more minutely, and to consider their mode of causation and diagnostic value.

Headache.

Of all the symptoms of intracranial tumour, headache is the most frequent; it is usually the first to attract attention, and is very frequently the symptom which is most distressing to the patient, and that which most urgently demands relief.

In the great majority of cases of intracranial tumour there is more or less headache. In some the pain is intense; the patient may describe it as "agonising," "unbearable," or may say he feels as if his "head would burst." The severity of the suffering may be so great as to suggest the idea of suicide.[1] As a result of this headache there is often sleeplessness and great exhaustion. In others, the headache is comparatively slight.[2] In a small proportion of cases it is altogether absent. In some cases the pain is dull and boring; in others, shooting and neuralgic in character. In many, the headache is paroxysmal or subject to paroxysmal exacerbations; in others, it is more or less constantly present; in some, and more especially (but not exclusively) in syphilitic cases, the pain is worse at night.

In many cases the paroxysms of headache are associated with vomiting or other signs of cerebral disturbance. In some cases, the patient is quite free from pain between the paroxysms. The length of the intervals is usually quite irregular; but in a few cases the

[1] See a case of glio-sarcoma of the cerebellum, reported by Dr. Leslie and myself, in the *Edinburgh Medical Journal* for January 1887, p. 591.

[2] See, for instance, a case of glioma reported by me in the *Edinburgh Medical Journal* for January 1887, p. 623.

paroxysmal exacerbations seem to be associated with menstruation, and to occur periodically every month.

Anything which deranges the cerebral circulation, such as hanging the head down, rising from the recumbent to the erect position, coughing, sneezing, laughing, straining at stool, &c., is apt to aggravate the headache, or to induce a paroxysm. In many cases the headache is felt when the patient first gets up in the morning.

The occurrence of headache and vomiting first thing in the morning is very suggestive of a cerebral lesion, but alcoholic dyspepsia and some other conditions, such as the vomiting of pregnancy, must be excluded.

The position of the pain is most variable. It is usually described as "internal," but in some cases is external and superficial. In some cases it is referred to the whole head; in others, it is limited to one side, to the vertex, or to the frontal, parietal, or occipital region. In a certain proportion of cases it is very limited and localised; in many of these, tenderness on skull percussion, or even on gentle pressure over the affected spot, is present. In some, localised alterations in the bones of the scalp (such as thickenings or depressions, syphilitic nodes, &c.) can be felt at the point of tenderness. In some cases, in which the pain is neuralgic in character, it is limited to the area of distribution of a particular branch of the fifth nerve.

The causation of headache in cases of intracranial tumour is probably various.

The most common cause is probably increased intracranial tension, with resulting stretching of the membranes and irritation of (both as a result of stretching of, and pressure on) the sensory nerve fibres distributed to the membranes of the brain and the cranial parietes. If this is so, we should naturally expect headache to be most prominent and severe in those cases in which greatly increased intracranial pressure is quickly produced (*i.e.*, in large, quickly growing tumours). When the increased intracranial pressure is slowly established, and more especially in those cases in which (in consequence of dropsy of the ventricles, a general œdematous condition or marked anæmia of the brain tissue) the cerebral sensibility is dulled, little or no headache may be complained of.

In some cases the headache is due to direct involvement of the

membranes, periosteum or bones, by the tumour, or by the inflammatory changes produced in the immediate neighbourhood of the new growth, with consequent pressure on, or inflammatory irritation of, the branches of the fifth nerve which are distributed to the affected part.

The effect which rapid alterations in the cerebral circulation, such as are produced by sudden rising from the recumbent to the erect position, sneezing, coughing, &c., may have in aggravating the headache has already been alluded to. *Vice versâ*, the relief which is in some cases afforded by free purgation, the application of cold to the head, and the administration of remedies which reduce the arterial blood pressure, will be afterwards described in more detail.

In some cases headache is due to direct implication of the trunk of the fifth nerve, or of some of its larger branches; and in such cases the pain is referred (in accordance with the law of eccentric projection) not so much to the seat of the lesion, as to the area of distribution of the affected nerve.

In some cases the headache of intracranial tumour is perhaps the result of a nerve storm, similar in character to that which produces the pain of ordinary sick headache or migraine. Such, at all events, was the late Dr. Hilton Fagge's view. He writes—"Now my hypothesis is that a tumour or tubercle causes a transitory vertigo, or an epileptiform attack, or an attack of sick headache, in exactly the same way as any other disturbing agent. I conceive that the nerve storm so produced has exactly the same effect as when it is merely the result of over-fatigue, or irritation of the generative organs, or disorder of the stomach. And I think it is probable that frontal headache, even when unattended with the other characteristic symptoms of an attack of migraine, is yet very often of that nature. If this be granted, it ought to follow that pain in the forehead should point less directly to the anterior part of the brain as the seat of a tumour, than occipital pain to the cerebellum or posterior lobes. I do not know whether growths situated in any particular region are more apt than others to be accompanied by vertigo or epileptiform convulsions, as distinguished from those seizures which are limited to the muscles of certain parts, and to which I shall presently refer. But Dr. Russell Reynolds was led, by the examination of a large number of cases, to the conclusion that convulsions in general were

less common when the disease affected the anterior lobes than when it occupied the posterior lobes or the cerebellum."[1]

The diagnostic value of headache as a symptom of intracranial tumour.—Headache is such a common symptom, and may be due to so many different conditions, both functional and organic, that *per se* it is not of much importance as an indication of serious organic cerebral disease (*e.g.*, tumour) unless it is very persistent and severe, and unless all the other conditions which may give rise to headache can be excluded.

But since it is the most frequent of all the symptoms of intracranial tumour, it is of great importance when associated with other general symptoms (vomiting, and more especially double optic neuritis) indicative of tumour, or when distinct indications of a localised cerebral lesion, such as localised spasms or paralysis, are present.

While it may be stated that in the absence of headache a *positive* diagnosis of intracranial tumour is seldom possible, it must be remembered that the absence of headache does not absolutely exclude tumour.

It must also be remembered that headache is often a prominent symptom in the very conditions which are most liable to be confounded with tumour—such as Bright's disease, plumbism, errors of refraction with anæmia, migraine, extracranial syphilis, meningitis, &c.

The localising value of headache is not as a rule great. Frontal headache more especially is apt to mislead the observer. In some cases, however, the pain corresponds more or less closely, and in a few cases most accurately, to the position of the tumour. In many subtentorial (cerebellar) tumours the pain is chiefly referred to the back of the head; and in tumours of one hemisphere it is not very uncommon to find the pain located on one (the same) side of the head. The localising value of headache is much increased if, in addition to the fact that the pain is confined to a limited area, there is also tenderness on percussion, or some other local alteration (such as a depression or swelling on the surface of the cranium) at the seat of the pain.

Localised pain, with tenderness on pressure, suggests very strongly that the tumour is superficial, and that the bone or membranes are affected. In syphilitic cases, in which the pain is often of this

[1] *Principles and Practice of Medicine*, vol. i., p. 526.

character, and indeed in all cases in which it is superficial and localised to one part of the cranial wall it may of course, be the result of an *extra*cranial as well as intracranial lesion.

In those cases in which the pain in the head is superficial and neuralgic in character, the fact that it is felt in the areas of distribution of *all* the branches of the fifth nerve makes it much more probable that it is due to central disease (*e.g.*, pressure on the nerve *within* the cavity of the cranium) than when one division only of the nerve is involved.

In illustration of the very definite manner in which pain and other abnormal sensations are sometimes limited to the exact seat of the intracranial tumour, the following statement of Hilton Fagge may be quoted :—"The museum of Guy's Hospital contains a large tumour, three inches in diameter, which I found many years ago in the left hemisphere of a girl, a patient of Dr. Wilks, who suffered severely from pain in the head, and who had declared that when she turned her head to one side she felt something move in its interior. Another preparation consists of a small growth from the dura mater, taken by Dr. Day, of St. Neots, from an old woman who died of bronchitis. She had often expressed a wish that her head should be opened, because for years she had experienced anomalous pains in it, and a sensation of coldness at one spot, not larger than a shilling ; this corresponded very nearly with the seat of the tumour, which was found after her death." [1]

The diagnostic value of headache in a pathological sense is not, as a rule, great.

When slight, or altogether absent, we expect the tumour to be stationary or of very slow growth. The reverse does not hold good, for a slow growing tumour may be attended with very severe headache. In a case of cerebellar tumour of several years duration, which I have reported in conjunction with Dr. Leslie, the headache was so severe as to suggest the idea of suicide to the patient; and, speaking generally, we infer, in cases of this description, that the tumour is simple (non-malignant), localised, and encapsuled, rather than malignant and diffused. Aneurisms on the large vessels at the base of the brain may even, when of considerable size, be unattended with headache.

[1] *Principles and Practice of Medicine*, vol. i., p. 328.

In many cystic tumours, and in many gliomata, the pain is very slight; but in other tumours of the same pathological structure the reverse is the case. In the *Edinburgh Medical Journal* for January 1887, pp. 591 and 623, I have reported two cases of sarco-glioma, with hyaloid degeneration, and the formation of large cysts. The pathological structure of the two tumours was identically the same; in both, the tumour had evidently been of slow formation and very slow growth. In one, in which the new growth was situated in the left frontal lobe, there was no increased intracranial tension (verified post mortem), and there had been no optic neuritis, very slight headache, little or no vomiting. In the other, in which the tumour involved the cerebellum, very great increase of the intracranial pressure was found after death, while during life agonising headache, very urgent vomiting, and intense optic neuritis were present. The difference in the symptoms in these two cases was probably due to the difference in the position of the tumour, and to the different effects which were produced as regards the intracranial pressure; for, as previously remarked, the pathological character of the tumour was in both cases identically the same.

In gliomatous tumour of the cerebrum itself headache is not infrequently absent; thus, in a hundred cases of cerebral tumour, analysed by Drs. Mills and Lloyd, it is stated that there was no headache in only five (no mention, however, being made of the presence or absence of headache in twenty-six). Three of these five were found to be gliomata; of the remaining two, one was probably gliomatous, and the other a cyst in the brain substance.[1] The fact that the pain is worse at night is very suggestive (though not pathognomonic) of the syphilitic character of the tumour.

In many of the cases in which the pain is localised to a particular part of the cranial wall, and associated with tenderness on pressure (skull percussion), the tumour is syphilitic or malignant in character.

Vomiting.

Next to headache and double optic neuritis, vomiting is probably the most common symptom of intracranial tumours. In some cases it is very severe and distressing. The great characteristic of cerebral

[1] Pepper's *System of Medicine*, vol. v., p. 1033.

vomiting is that it is purposeless—*i.e.*, that it occurs without obvious cause, at irregular intervals, and that it does not necessarily have any fixed relationship to the ingestion of food or drink. Cerebral vomiting is often, though by no means always, associated with a clean tongue. It may be unattended with nausea, but is often followed by marked exhaustion. In many cases it is paroxysmal, and is associated with exacerbations of the headache. Like headache, it is often apparently due to disturbance of the cerebral circulation; and frequently occurs before breakfast, when the patient first rises from the recumbent to the erect position.

The causation of vomiting in cases of cerebral tumour.—Like headache, vomiting is in many cases, I believe, due to the irritation of the sensory nerve fibres in the cerebral membranes—the result either of increased intracranial pressure, or of direct involvement by the new, growth or by the inflammatory changes in its neighbourhood. Possibly in some cases vomiting may be caused reflexly by irritation originating in the tissues of the brain itself.

The effect which derangements of the cerebral circulation have in producing vomiting, and Dr. Hilton Fagge's views as to the relationship of the vomiting in cases of cerebral tumour and in migraine, have been already alluded to.

Direct irritation of the "vomiting centre" in the medulla oblongata, by a tumour in its immediate neighbourhood, is another way in which very severe and obstinate vomiting may be produced.

It is also probable that in some cases of cerebral tumour, in which there is severe vertigo, vomiting may be due to an "overflow" of irritation from the vertiginous to the vomiting centre.

The diagnostic value of vomiting in cases of cerebral tumour.—Although vomiting is a very frequent symptom in cases of intracranial tumour, it is *per se* of little diagnostic value, for so many other different conditions may produce it. In estimating the value of vomiting as a sign of intracranial tumour, it is of course necessary to exclude derangements of the stomach and other abdominal causes; and to satisfy oneself that the vomiting is cerebral. It is also necessary to remember that vomiting is often a prominent symptom in many of the conditions which most closely resemble intracranial tumour, such as meningitis, Bright's disease, plumbism, migraine,

hysteria. Care, too, must be taken not to confound the morning sickness due to alcoholismus, or pregnancy, with that due to an intracranial tumour.

As a *localising* symptom, and as an indication of the pathological character of the new growth, vomiting is not of much value. The greater the cerebral and meningeal irritation, the greater, as a rule, the vomiting. Those tumours, therefore, which grow rapidly, which produce rapid increase of the intracranial pressure, and which cause meningitis (such as syphilitic, tubercular, and malignant tumours), are, other things being equal, the most likely to produce vomiting.

Tumours which are situated far forward (tumours of the frontal lobe, for example) seem to be less frequently associated with severe vomiting than those which are situated far back (subtentorial tumours, for instance). In short, the nearer the tumour is to the "vomiting centre," the greater is the tendency, other things being equal, to the production of vomiting. Very severe and very obstinate vomiting is therefore suggestive of a lesion in the neighbourhood of the pons Varolii or medulla oblongata. There are, however, so many sources of fallacy in connection with this point, that the localising value of vomiting is not great.

Vertigo.

This is another very common symptom in cases of intracranial tumour. It is usually slight and transitory, but is sometimes both persistent and severe. In many cases the giddiness is produced or aggravated by suddenly rising from the recumbent to the erect position, stooping the head forward, &c. In most cases the patient simply feels giddy; in some, he feels as if surrounding objects were moving in some particular direction. In a few cases the vertigo is "active," and the patient feels as if he himself were being moved or whirled through space. In one of my cases, in which vertigo was very distressing, the patient stated that the ceiling seemed to be constantly falling down upon the top of her. In that case, the tumour—a large secondary cancerous nodule (see Fig. 7)—was situated in the right lobe of the cerebellum.

Tumours of the middle lobe of the cerebellum are frequently attended with severe vertigo. This is, however, by no means

always so; see, for example, the case of melanotic sarcoma, which I have reported in conjunction with Dr. Allan Jamieson, in the *Edinburgh Medical Journal* for July 1887, and which is represented in Fig. 8.

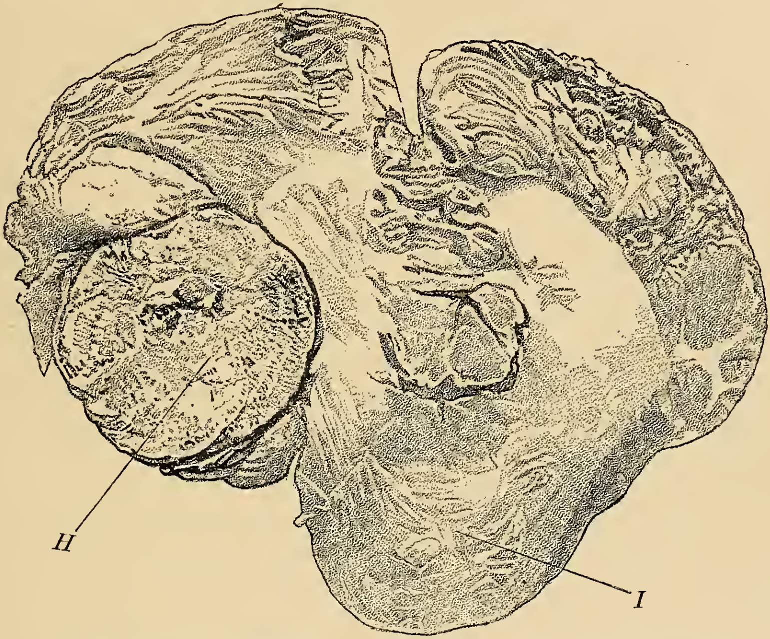

Fig. 7.—Transverse section through the cerebellum and pons Varolii, showing a large nodule of secondary cancer in the right lateral lobe of the cerebellum. The letter *H* points to the tumour; *I*, to the pons Varolii.
The right middle peduncle of the cerebellum was compressed by the new growth. (The case is reported in *Brain*, January 1888, page 503.)

The causes of vertigo in cases of cerebral tumour are various. In many cases it is probably due to temporary derangements of the cerebral circulation; in others it is probably the result of irritation of the cerebral membranes, caused by increased intracranial pressure, meningitis, &c.; in some it is perhaps due to irritation originating in the brain tissue itself. In a few cases—and these are the cases in which the vertigo is most distressing and severe—it is due to direct irritation of some part (of the intracranial portion) of the mechanism concerned in the regulation of the head to the plummet line of the body. Tumours which involve the cerebellum or medulla oblongata, or which press upon the eighth nerve (portio mollis of the seventh

nerve), may probably produce giddiness in this way. Drs. Mills and Lloyd also suppose that vertigo may be due to pressure on the labyrinth. They say—"Experiments have demonstrated the existence of a communication between the arachnoid cavity and the labyrinth, and consequently the increased intracranial tension present may result in pressure on the labyrinthine fibres of the auditory nerve, and thus occasion vertigo."[1] In some cases the vertigo is probably ocular. A cerebral tumour may cause paralysis of some one or other of the muscles of the eyeball, in consequence of which ocular vertigo is developed. As we might *a priori* suppose, this form of giddiness is not common, even in those cases in which a cerebral tumour does produce an ocular paralysis; for ocular vertigo is chiefly met with as the result of a quickly established (recent) ocular paralysis. In those cases in which the paralysis is slowly established or of long duration—as it is in many cases of ocular paralysis due to cerebral tumour—the nerve centres accommodate themselves to the double vision, and the vertigo disappears, or is not produced.

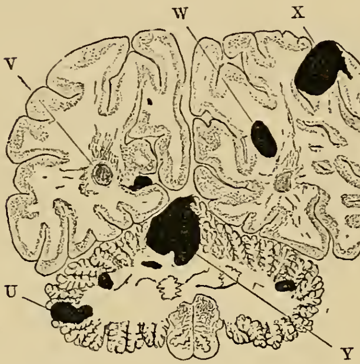

FIG. 8. — Transverse vertical section through the brain in a case of melanotic sarcoma, showing numerous melanotic deposits both in the brain and cerebellum. (Reduced from a photograph.)

The letter *U* points to a nodule of some size in the left lateral lobe of the cerebellum; *V*, to the left lateral verticle; *W* and *X* to large nodules in the right cerebral hemisphere; *Y* to a large nodule in the middle lobe of the cerebellum.

Vertigo was never complained of during the whole course of the case, which is reported in the *Edinburgh Medical Journal* for July 1887.

The diagnostic value of vertigo in cases of cerebral tumour is *per se* not great. It is necessary, of course, to exclude all the other possible causes of giddiness, more especially Ménière's disease, in which many of the symptoms are identical with those of cerebellar tumour; and it is only when other symptoms of tumour are present that the vertigo can with any degree of confidence be attributed to this cause. Very severe vertigo, in combination with other symptoms indicative

[1] Pepper's *System of Medicine*, vol. v., p. 1035.

of a tumour, suggests that the new growth is situated in the cerebellum (possibly in the middle lobe); or that it involves the intracranial portion of the eighth nerve (portio mollis). In the latter case, marked unilateral deafness, from involvement of the auditory fibres, and very probably facial paralysis, from implication of the portio dura or seventh nerve, would probably be present.

Marked vertigo, unilateral deafness, and facial paralysis, when there is no apparent lesion of the ear—when associated with other symptoms of an intracranial tumour—are indicative of a tumour at the base.

CHAPTER III.

CHANGES IN THE FUNDUS OCULI—DOUBLE OPTIC NEURITIS AND OPTIC ATROPHY.

Of all the symptoms of intracranial tumour, double optic neuritis is the most important; *firstly*, because it is an objective sign, which does not depend upon the mere sensations and statements of the patient, but which is attended with distinct physical alterations which can be seen by the physician; *secondly*, because it is present in the great majority of cases of intracranial tumour *at some period or other of their course;* and *thirdly*, because, unlike headache, vomiting, and vertigo, it is a symptom which is not commonly produced by other conditions; or, to state the matter more accurately, an intracranial tumour is by far the most common condition which is associated with double optic neuritis.

The degree of change in the optic discs may vary from mere engorgement of the veins, without swelling of the disc or blurring of its edges, up to the most intense neuritis.

In the vast majority of cases the optic neuritis associated with intracranial tumours is bilateral, though it very frequently happens that the changes are more marked in one eye than in the other. Cases are occasionally met with in which the neuritis is confined to one disc. Dr. Hughlings Jackson thinks that, in cases of this description, the disc on the opposite side to the brain lesion is more frequently affected than that on the same side; but whether this is more than accidental, and whether it is a point of any importance, is perhaps doubtful. The number of cases of intracranial tumour in which the papillitis has been unilateral is, in my opinion, too small to allow of any very definite generalisation being made.

In Figs. 9, 10, and 11 the appearances of the fundus oculi in health and in papillitis are represented.

After an inflammatory condition of the discs has lasted some time,

vision is usually more or less affected ; and in many cases, even if the optic neuritis should subside and completely disappear, some atrophy of the optic discs, with more or less impairment of vision, remains.

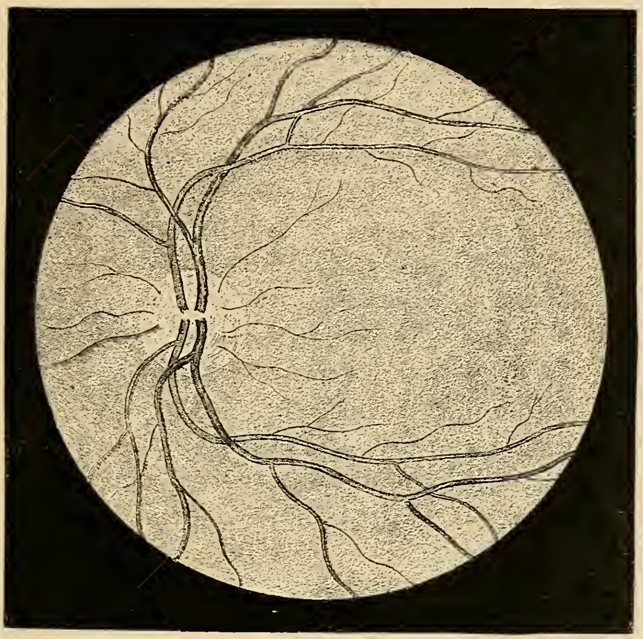

Fig. 9.—The normal fundus oculi. (Indirect image.)

But while vision is usually affected sooner or later, and to a greater or less degree, in long-continued optic neuritis, it is of the utmost importance to remember that it (both the acuity of vision and the extent of the visual field) may be absolutely normal, even when marked double optic neuritis can be seen with the ophthalmoscope. One of the most striking cases of this kind which have come under my observation was that of a man aged twenty-three, who was recently sent to me by Dr. Berry, suffering from well-marked double optic neuritis of some weeks' duration. The exact causation was obscure ; he had apparently had an attack of meningitis, characterised by headache, vomiting, and elevation of temperature ; there was some double vision, but there were no symptoms which enabled me to diagnose positively an intracranial tumour, and a guarded opinion was given. The double optic neuritis, which was very marked, and

which is represented in Fig. 11, lasted for many weeks, but ultimately completely disappeared under the continued administration of large doses of iodide of potassium. The acuity and the field of vision, which were regularly examined, were never in the least degree impaired; both at the height of the condition and after all inflammatory changes had disappeared they remained absolutely normal. The patient had not had syphilis.

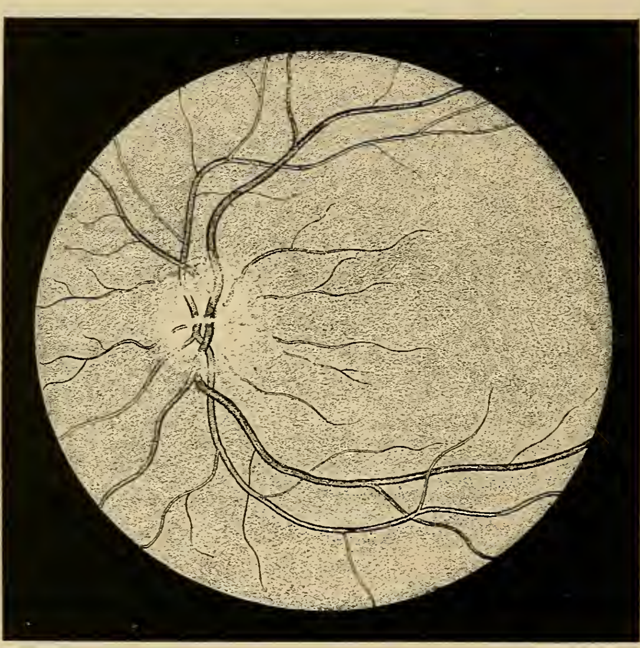

FIG. 10.—Fundus oculi in a case of intracranial tumour, showing well-marked papillitis. (Indirect image.)
The disc is markedly swollen, and its edges ill defined. The vessels are obscured, more especially where they pass over the edge of the swollen disc.

I have reported several cases of cerebral tumour, one of which is represented in Figs. 1, 2, 3, and 4 (see pages 11-14), in which well-marked optic neuritis was associated with good or perfect central vision; and this fact, which was long ago insisted upon by Dr. Hughlings Jackson, is now widely recognised by all competent authorities. In reference to this point Dr. Stephen Mackenzie states that "he would go so far as to say that in the practice of physicians who examined *all their cases* with the ophthalmoscope, whether the

case was a cerebral one or otherwise, at least in one-half, if not more, of the cases in which optic neuritis was discovered it would be found unassociated with any marked, and often without apparent appreciable

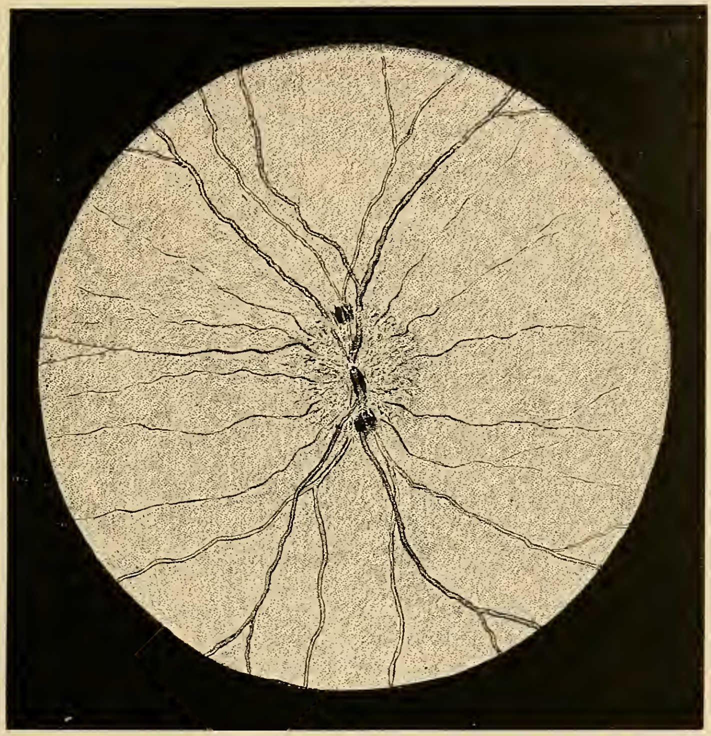

FIG. 11.—Fundus oculi in a case of well-marked papillitis. (Indirect image.)

Both the arteries and veins are obscured as they pass over the edge of the swollen disc. Two hæmorrhages are seen on the disc ; several minute hæmorrhages could be seen on the swollen disc by the direct method of examination.

The veins are drawn rather too small.

defect of sight,"[1] while Dr. Buzzard stated in the same debate that "he continually found—indeed, he thought it was the rule—that persons in whom the optic discs were greatly swollen not only had no complaint to make of their sight, but could read the smallest type."[2]

[1] *Transactions of the Ophthalmological Society of the United Kingdom*, vol. i., p. 97.
[2] *Ibid.*, p. 97.

The fact that there may be little or no defect of vision when there is well-marked optic neuritis, shows the fallacy of trusting to the condition of vision as an index of the state of the fundus oculi, and teaches the necessity of the routine use of the ophthalmoscope. Whenever, in short, there is any reason to suspect intracranial disease —more especially tumour—the fundus oculi should be examined with the ophthalmoscope. It is not too much to say that the ophthalmoscope is quite as important in the diagnosis of some nervous affections, as the stethoscope is in the diagnosis of some cardiac and respiratory diseases. The routine use of the one is just as necessary, if mistakes are to be avoided, as the routine use of the other; for just as some serious cardiac affections—aortic regurgitation, for example—may be unattended by symptoms, but readily detected on stethoscopic examination, so the double optic neuritis, which is easily enough seen in most cases by means of the ophthalmoscope, may be unattended with any alteration of the visual acuteness, or indeed any limitation of the field of vision.

In the majority of cases the optic neuritis commences gradually and continues for a long time. As a rule, too, the dimness of vision due to optic neuritis, or resulting optic atrophy, is slowly and gradually established. Cases are, however, met with in which total loss of vision occurs suddenly. I have reported a case of cerebellar tumour with extensive ventricular dropsy, in which sight and hearing were both quickly lost; and Dr. Hilton Fagge mentions a case in which sudden loss of vision also occurred.

Double optic neuritis is essentially a "*general*" and not a localising symptom; for it may result from tumours in any part of the cranial cavity. In the very few cases of cerebral tumour in which the optic neuritis is confined to one eye, the tumour has been found in the opposite cerebral hemisphere. Unilateral optic neuritis (*i.e.*, neuritis confined throughout the course of the case to one eye), is, however, strongly suggestive of local disease in the eyeball or orbit, or of localised pressure on the optic nerve in front of the optic chiasma, rather than of an intracranial tumour.

In estimating *the diagnostic value of double optic neuritis as a sign of intracranial tumour,* it must be remembered—

Firstly. That in a certain proportion of cases of intracranial tumour double optic neuritis is never observed throughout the whole

course of the case; and that in others it has either not developed, or has already subsided, when the case comes under observation. Hence the absence of double optic neuritis does not necessarily exclude the presence of a tumour. The fact that there is no double optic neuritis does, however, suggest doubt; and unless the other symptoms of tumour are very clearly defined, or unless the physician feels satisfied that there is no other condition except tumour which could reasonably be expected to account for the phenomena of the case, he will be wise, in the absence of double optic neuritis, to hesitate before committing himself to a positive diagnosis.

In those cases of intracranial tumour—and I agree with Dr. Buzzard in thinking that they are mostly syphilitic—in which the double optic neuritis subsides under treatment, it is usually, but not always, possible to say from the condition of vision and the state of the fundus that the optic discs have been previously inflamed.

In some cases, more especially when the optic neuritis is recognised and vigorously treated in its earlier stages, recovery, both as regards vision and ophthalmoscopic appearances, may be perfect. In others, some dimness of vision, some limitation of the fields, or some changes in the fundus, such as white lines along the course of the vessels or white patches near the edge of the disc, remain. In other cases, again, marked defects in vision and striking ophthalmoscopic appearances remain behind.

The recognition of any changes in the fundus indicative of past neuritis is in some cases of great diagnostic importance.

Secondly. That double optic neuritis may occur in some of the conditions, which are most easily mistaken for intracranial tumour, and in which the difficulties of diagnosis are sometimes very great. Double optic neuritis is, for example, almost always present in lead encephalopathy; it is not uncommon in meningitis and cerebral abscess; and it may occur in Bright's disease. It has occasionally been observed in association with uterine or ovarian derangements, hypermetropia, and anæmia, which, when complicated with hysterical or other nervous symptoms, may under such circumstances (*i.e.*, when associated with double optic neuritis) be easily mistaken for cases of intracranial tumour.

But while double optic neuritis is unquestionably of the greatest

diagnostic value as a sign of intracranial tumour, it must be remembered that, even when associated with the other typical (general) symptoms of cerebral tumour, and even with symptoms indicative of a local lesion, a tumour, properly so-called, is not always found after death. Fortunately for diagnosis, cases of this kind are quite exceptional and rare. The following, which occurred in the practice of Dr. Hughlings Jackson, and which is quoted by Dr. Hilton Fagge, is a case in point:—A woman, aged thirty-four, had for about a year been subject to attacks of severe headache accompanied with vomiting; for three months she had been blind. Her illness began with vertigo and momentary unconsciousness, after which she had headache for four days. At another time the pain lasted for three weeks. She was admitted into hospital, under Dr. Hughlings Jackson's care, on December 19th, 1874. On January 6th, 1875, she had an attack of pain so intense as to make her toss her head from side to side, holding it with her hands and saying, "Oh, my head," "I don't know what I shall do." She retched and vomited frequently. Both optic discs were greatly swollen, and the veins in them were dilated and tortuous. After the 10th, she sank gradually into what appeared to be natural sleep, which, however, passed into coma, and on the 12th she died by failure of the respiration. A tumour or some other similar lesion was confidently anticipated, but Dr. Sutton, who made the autopsy, found only certain microscopical changes in the substance of the cortex.

In the debate on optic neuritis which took place at the Ophthalmological Society a few years ago, Dr. Hughlings Jackson stated—"It is to be particularly mentioned that optic neuritis is found in some cases—clearly, from the symptoms, cases of intracranial disease—where ordinary examination (post mortem) discloses no local disease within the cranium or in any other part of the body." [1]

Dr. Stephen Mackenzie has published a case in which optic neuritis occurred in connection with what, at the post mortem examination, appeared to be simple atrophy of the brain; but a microscopical examination of the cortex of the brain showed a diffused cerebritis and inflammation extending down the optic nerve. There was no meningitis and no marked effusion into the optic sheaths. [2]

[1] *Transactions of the Ophthalmological Society of the United Kingdom*, vol. i., p. 61.
[2] *Brain*, vol. vii., p. 257.

The number of cases of intracranial tumour in which the neuritis has been observed on one side only is too small, in my opinion, to admit of any very definite generalisation being made.

Optic Atrophy.—This condition is sometimes met with in long-continued cases of intracranial tumour. It usually results from previous optic neuritis; but in a few cases it is primary, and may then be due to the pressure of a tumour, or of the dilated cerebral ventricles, upon the optic chiasma or optic nerve trunk.

In some cases of optic atrophy the patient is completely blind; in most, both the acuity of vision and the visual field are markedly impaired. When the atrophic changes are very slight—but in these it is difficult or impossible to recognise any distinct alterations in the fundus oculi by means of the ophthalmoscope—the acuity of vision (central vision) may be little if at all affected, while the visual field is more or less contracted. Colour vision is usually impaired. In post-neuritic cases the contraction of the visual field is often irregular in outline.

Now, since primary atrophy is very rarely indeed due to an intracranial tumour, while post-neuritic atrophy is common, it is a point of some diagnostic importance to be able to distinguish the two conditions. There are cases in which the distinction is impossible; but in the majority of cases in which the atrophy is post-neuritic, some indications or remains of the previous inflammation are to be seen. Indeed, in many cases the inflammatory changes (swelling of disc, covering up of the vessels, &c.) can still be recognised when the case comes under observation. But even in long-continued cases, and after all inflammatory changes have subsided, the appearance which the disc presents enables us in many cases to say that the optic atrophy is post-inflammatory and not primary. Opaque, chalky whiteness of the disc, irregularity or indistinctness of its margins, covering up of the origin of the vessels, the presence of white lines along the sides of the vessels, or of white patches on the surrounding retina, and the fact that the size of the vessels is markedly different from the normal, are in favour of the post-inflammatory character of the atrophy.

The Causation of the Double Optic Neuritis associated with Intracranial Tumours.

The causation of the double optic neuritis, which is so frequently associated with intracranial tumours, has given rise to much debate.

Before considering the different theories which have been from time to time advanced to account for its production, it may perhaps be well to lay down certain general propositions, with which most observers will probably agree. They are as follows:—

1. Double optic neuritis is present at some period or other of the course of the great majority of cases of intracranial tumour.

My own experience with regard to this frequency is very much in accord with that of Gowers, who states that double optic neuritis is present in about eighty per cent. of the cases of intracranial tumour, at some period or other of their course.

2. It is difficult or impossible in many cases to distinguish the papillitis which is due to choking of the disc, from that which is due to descending neuritis.

Most observers are now agreed that the two conditions are different degrees of one and the same process; in other words, that the so-called "choked" disc is the result of a true inflammatory process, and is not a mere œdema, the result of mechanical compression.

It is probable, however, that the papillitis which is associated with cerebral meningitis (and which is presumably, therefore, a typical descending neuritis) may, in some instances at all events, be distinguished in its early stages from the papillitis which is due to tumour. With reference to this point Dr. Gowers says—"Apart from the distinguishing characters afforded by the degree of neuritis, I have only noted one difference, viz., that during the early stage of inflammation, when it is coming on and the swelling is distinct, the papilla is paler in meningitis than in tumour."[1]

It has appeared to me that the papillitis of tumour is more frequently preceded by dilatation of the retinal veins than the retinitis due to meningitis. Gowers, however, with his much larger experience, has come to a different conclusion. He states—"In tubercular meningitis Garlick's careful observations have shown that, while papillitis is

[1] *Transactions of the Ophthalmological Society of the United Kingdom*, vol. i., p. 104.

not, fulness of the veins is, related to an increased intracranial pressure. In the course of a descending neuritis, the distention of veins may be very great, as I have several times observed, both during life and after death. In tumour the veins at first, and often throughout—when the neuritis does not reach a considerable degree of intensity—are little above the normal size, and present no tortuosities except those which are given them by the prominence of the papilla. The great distention of the veins and narrowing of the arteries occur when the inflammation has reached a certain degree of intensity: this points to the neurotic process in the papilla as causing strangulation by pressure in, and constriction of, the vessels."[1]

The papillitis associated with tumour is as a rule more intense than that due to meningitis.

3. The double optic neuritis associated with tumour is a "general" and not a "localising" symptom; in other words, it may be due to a tumour in any part of the cranial cavity. It may result from a tumour either in the substance or on the surface of the cerebrum, cerebellum, or pons Varolii. It is, however, a question whether double optic neuritis is not more frequently associated with tumours in some situations than in others. Thus Drs. Walter Edmunds and I. B. Lawford state that in an analysis of ninety-six cases of fatal cerebral tumour, they found optic neuritis was present in eighty-six per cent. of the cases in which the new growth was situated in the basal ganglia or in the cerebellum, but in only forty-six per cent. of those cases in which it was seated at the convexity. In my own experience, tumours of the convexity are more frequently associated with double optic neuritis than these statistics show; but I am quite prepared to allow that tumours in the basal ganglia and cerebellum are more likely to be associated with papillitis than tumours of the convexity, in consequence, I think, of the fact that they more frequently produce ventricular dropsy, great increase of the intracranial pressure, and their results.

Perhaps tumours of the medulla oblongata are less frequently attended with double optic neuritis than tumours in other parts.

With regard to this point Dr. Hughlings Jackson says—"Like Gowers, I have not yet seen optic neuritis with tumour of the

[1] *Medical Ophthalmoscopy*, Second Edition, p. 71.

medulla oblongata;" but he adds, "I should say that I have only once seen tumour limited to that part, and forget whether there was neuritis or not." [1]

If it is the case that tumours of the medulla oblongata are less frequently associated with double optic neuritis than tumours in other parts of the intracranial cavity, the fact may perhaps be explained by supposing that tumours in this situation seldom attain to any great size, and that they are, in some instances at all events, unattended with ventricular dropsy or any great increase of the intracranial pressure.

4. Double optic neuritis may occur in connection with any form of new growth. It is associated with small as well as with large tumours, and may be associated with almost any (pathological) variety of tumour.

This proposition must, however, be qualified by stating that slow-growing tumours, which neither produce increased intracranial pressure nor irritation of the intracranial contents, may be unattended with optic neuritis, or, indeed, with any other symptoms. Small psammomata, for instance, are as a rule completely latent; and even tumours of a larger size, such as that which was found in the case of the late Professor John Hughes Bennett, may, if they fulfil the conditions stated above, produce no disturbance.

Vice versâ, the tumours which do most frequently produce double optic neuritis are those which cause either increased intracranial pressure, or irritation of intracranial structures (meningitis or cerebritis), or both.

Presumably, therefore, in one or other, or all of these conditions, the cause of the double optic neuritis, which is associated with intracranial tumours, is to be found; and, as a matter of fact, these three pathological alterations form the basis of three of the theories which have been advanced to explain its causation, viz.:—

(1.) The increased pressure theory.

(2.) The descending neuritis theory.

(3.) The vaso-motor irritation theory.

A fourth theory is that which supposes that some irritant, produced in or around the tumour, becomes mingled, as it were, with

[1] *Transactions of the Ophthalmological Society of the United Kingdom*, vol. i., p. 79.

the cerebro-spinal fluid, and being conveyed by it into the subvaginal space which surrounds the orbital portion of the optic nerve, produces irritation and inflammation of the nerve—in other words, double optic neuritis.

Leber, who first advanced this theory, supposed that "intra-cranial tumours, as also tuberculosis, give rise to congestion of the vessels, secretory inflammation, hydrops ventriculorum, and increased pressure; and that the products of tissue-change of those neoplasms, which become mingled with the inflammatory transudations, act as an inflammatory stimulus, and, passing with the cerebro-spinal fluid into the intervaginal space of the optic nerve, as far as the eye, give rise there to neuritis and papillitis."[1]

Deutschmann, who adopts Leber's theory, suggests that the irritating elements are perhaps micro-organisms, which, being already present in the system, congregate around the tumour as a "place of least resistance," and are thence carried to the optic nerves in the manner described above.

This theory, which supposes both the presence of an irritant in the cerebro-spinal fluid and increased intracranial pressure (for Deutschmann admits that the increased intracranial pressure is a favouring, though he thinks it is not an essential condition), may be termed the *irritation-pressure theory*.

I may at once say that, for my own part, I think it probable that the double optic neuritis of intracranial tumours is not always produced in the same manner.

It must, I think, be admitted that papillitis may be due to a *descending* inflammation passing from the brain along the nerve, or along the meninges (connective tissue and blood-vessels) to the nerve; but that this is the manner in which it is usually produced in cases of tumour is, I think, by no means proved.

The irritation-pressure theory of Leber and Deutschmann seems to me the most plausible which has yet been advanced; but before it can be definitely accepted, the presence of an irritant and its exact nature require, I think, further demonstration.

While the experiments of Deutschmann would appear to show that increased intracranial pressure *per se* is not the only or the

[1] *Transactions of the International Medical Congress*, 1881, vol. iii., p. 57.

essential cause of papillitis, I am, for the reasons which will be presently given, of opinion that it is a factor which cannot be ignored; and that, in many instances at all events, it does play an important, though a secondary, part in the production of papillitis.

Before considering the different theories in detail, it may perhaps be well to briefly summarise the chief post mortem appearances in cases of intracranial tumour which have a direct bearing upon the question under discussion.

In the great majority of cases there is evidence of increased intracranial pressure; flattening of the convolutions, effacement of the sulci, and in many cases a scanty amount of sub-arachnoid fluid.

Dropsical distention of the ventricles is frequent.

In many cases, localised meningitis, cerebritis, or softening is present in the immediate neighbourhood of the tumour; and, in a few cases, there is evidence of localised meningitis in other parts (*i.e.*, in parts at a distance from the tumour, and which have no apparent connection with the new growth). Except in tubercular cases, generalised meningitis is rarely found.

The vaginal sheaths surrounding the optic nerves are, in the majority of cases, distended with fluid, or, if collapsed and undistended, appear, from their flaccid and loose condition, to have been over-distended.

In many instances, in which microscopic examination shows distinct evidence of inflammatory changes in the papilla, in the optic nerve behind the eyeball, and in the fibrous coverings of the nerve, these structures appear normal to the naked eye. This is a point of some practical importance, and I would emphasise the fact that in some cases, in which well-marked optic neuritis was present during life, I have been unable, even with the aid of a pocket lens, to satisfy myself that the optic papilla was distinctly abnormal. In my experience, microscopical examination is, in some instances, required to decide the point.

For the same reason, the fact that there is no naked-eye evidence of meningitis within the cranial cavity is not perhaps sufficient to exclude cerebral meningitis. It is possible that in some cases of intracranial tumour, in which the naked-eye appearances are simply those

of increased pressure, the microscope may show some evidences of inflammatory changes in the meninges. I cannot, however, say that my own observation lends any definite support to this view.

In this connection it is important to note that in many cases of intracranial tumour the microscope shows diffuse irritative changes (in the form of proliferation and enlargement of the connective tissue elements—Deiter's corpuscles), not only in the immediate neighbourhood of the tumour, but more or less widely diffused throughout the swollen brain tissue. Whether, as has been suggested, this diffuse irritative change is a cause, or the cause, of the optic neuritis, headache, and other general (non-localising) symptoms of intracranial tumours, is a point which has not yet been definitely determined. But it is important to note that cases have been reported by Hughlings Jackson, Stephen Mackenzie, and Gowers, in which optic neuritis was associated with headache, vomiting, and all the typical (general) symptoms of "coarse" intracranial disease, and in which an atrophied condition of the brain, with microscopic appearances indicative of a diffuse irritative change in the nerve centres, but no tumour or other "coarse" localised lesion, was found post mortem.

With regard to the microscopical appearances in the optic nerve itself, I regret that I am unable to speak as definitely as I would wish. Although I have examined a large number of optic nerves, both in cases of intracranial tumour and in other conditions, I have failed to satisfy myself with regard to many of the points at issue. This is partly due to the fact that the inherent difficulties connected with the subject have seemed to me to be greater than some writers appear to have found them; and partly to the circumstance that in most instances my examination has been limited to the intra-orbital nerve, and that I have not examined the intracranial portion and the optic chiasma, the condition of which has an important bearing on some of the points in dispute. Possibly, too, the methods of preparation which I have employed (hardening in Müller's fluid, freezing, staining with picro-carmine or borax-carmine, and mounting in Farrant's solution, or in xylol balsam) are not the most satisfactory for bringing out the morbid appearances, but they are those with which I am most familiar, and with which I was in the habit of working when, as Pathologist to the Edinburgh Royal Infirmary,

material of this kind came in my way. Embedding in celluloidin is perhaps preferable to the freezing method.

FIG. 12.—Camera lucida drawing of a section through the optic nerve and surrounding parts in a case of glioma of the cerebellum, showing well-marked papillitis. There is great swelling and vascularity of the optic disc, the presence of inflammatory products in the subvaginal space, and swelling of the connective tissue trabeculæ surrounding the pial sheath. (From a preparation stained with picro-carmine, cleared with absolute alcohol and oil of cloves, and mounted in xylol balsam.)

The letter *C* points to the centre of the optic papilla; *D* to a large vessel which is distended with a dense mass of blood corpuscles; *E* to the swollen deeper layers of the retina adjacent to the disc; *F* to the deeper layers of the retina where they are healthy; *G* to the choroid, which has become detached from the retina and sclerotic; *J* to the dural sheath of the optic nerve; *K* to the subvaginal space, which is distended with inflammatory products right up to its termination, *L*.

The case is reported in the *Edinburgh Medical Journal*, January 1887, p. 591.

In well-marked cases of optic neuritis, the microscopic appearances in the papilla itself (swelling of the nerve fibres, distention of the

capillaries in the disc and of the large vessels of the adjacent retina, abundant infiltration with leucocytes, and in some instances extravasations of red blood corpuscles and even hæmorrhages of some size in the disc itself, or in the surrounding retina; with, in some cases, swelling of the nerve cells of the adjacent portions of the retina) are quite distinctive of inflammation. (See Fig. 12.)

In such cases I have, like Leber and Deutschmann, usually found nuclear proliferation and engorgement of the blood-vessels in the adjacent portions of the choroid.

In most cases of well-marked papillitis which I have examined, there has been distinct nuclear proliferation in the inner and outer sheath of the nerve, the presence of corpuscular elements (leucocytes, and, in a few instances, red blood corpuscles) in the subvaginal space, together with what has appeared to me to be swelling of the connective tissue trabeculæ, which pass across the vaginal space and connect the outer with the inner sheath. I have considered these appearances, when well marked, as indicative of inflammation (perineuritis), and have regarded them as being in all probability the cause of the papillitis. But whether they were due to a process of "descending" inflammation, or whether they were caused by an irritant being forced by the increased intracranial pressure into the subvaginal space (and this is one of the weak points in my observations), I have been unable to decide.

In this connection I may state that I have in several instances observed a minor degree of the same changes in cases in which there was neither intracranial tumour nor optic neuritis; and I am, in consequence, disposed to think that nuclear proliferation in the outer and inner sheaths of the nerve and swelling of the connective tissue trabeculæ may be more frequent, and consequently of less importance as a sign of "descending" inflammation, than some observers appear to think.

In one case of chronic Bright's disease (the cirrhotic form) in which the microscopical examination showed distinct evidence of papillitis, the same inflammatory appearances in the sheaths of the nerve and subvaginal space were well-marked. This is a point of interest when taken in connection with the following statement which Dr. Broadbent made in the discussion at the Ophthalmological Society on optic

neuritis. In speaking in support of the increased pressure theory, he stated—"A fact of great weight, which had only come under his observation comparatively recently, was that, in the optic neuritis of renal disease, the same distention of the optic sheath was at times observed as in the intracranial disease. Now, besides the headache, there were other indications, such as the relief of uræmic convulsions by bleeding, that in kidney disease there was increased intracranial pressure—due, no doubt, to high arterial tension—and there was no clue to the occurrence of optic changes in one case of kidney disease and its absence in another, unless it were to be found in the fact that he had never yet seen them where the arterial tension was not excessive."[1]

In well-marked cases of papillitis the connective tissue trabeculæ and the nerve fibres of the lamina cribrosa have appeared to me to be swollen, and I am unable to speak in the same positive manner that Gowers does as to the absence of all constriction of the vessels at this point, although I quite admit that in most cases the constriction is seated in the swollen papilla. Gowers says, "I have never been able to discover any evidence of constriction of the vessels in the sclerotic ring or behind it. Their calibre here is always uniform. This statement is based on the most careful search for any evidence of such compression in a number of cases of papillitis from various intracranial diseases. In one case only was there an appearance of narrowing, and in this, from the unaltered course of an adjacent vessel, it was evidently due to a slight alteration in the position of the vessel at this spot, in consequence of which the sections ceased to pass through its widest part. It is always in front of the sclerotic, in the substance of the swollen papilla, that the vessels present conspicuous constriction, and are pressed upon and have their walls thickened by new tissue. Further, the most intense strangulation may occur in cases in which there is reason to believe that there is no intracranial disease; and in case 27, in which there was no intracranial condition that could cause any mechanical effect, the intra-ocular signs of constriction and mechanical congestion were very marked."[2]

[1] *Transactions of the Ophthalmological Society of the United Kingdom*, vol. i., p. 110.
[2] *Medical Ophthalmoscopy*, Second Edition, p. 71.

With regard to the condition of the optic nerve behind the papilla in cases of well-marked papillitis, I have in some instances found thickening of the connective tissue trabeculæ and excess of nuclei, more especially in those portions of the nerve adjacent to the sheath. In some cases these appearances have been slight, in fact not much greater than I have found in other cases in which there was neither intracranial tumour nor papillitis. And in this connection I would ask, what degree of trabecular thickening and nuclear proliferation is indicative of inflammation (neuritis)? When these appearances are little marked, I have felt considerable difficulty in deciding that they were distinctly inflammatory. In several instances, in which there was no papillitis, and no reason to suspect inflammatory change in the nerve trunk behind the papilla, I have found a degree of trabecular thickening and nuclear excess which has appeared to me to be excessive, and which would, I suspect, by some observers (if they were examining cases of papillitis) be considered as indicative of a descending neuritis.

One of the most typical cases of inflammation of the optic nerve behind the papilla which has come under my observation was a case of myxœdema. In that case, bright staining nuclei (leucocytes) were thickly scattered throughout the whole thickness of the nerve, and the minute blood-vessels in the trunk of the nerve behind the eyeball were dilated and engorged with blood. In no cases of papillitis due to intracranial tumour have I seen such intense neuritis in the post-ocular portion of the nerve as in this case.

Let us now briefly consider these different theories more in detail, and in considering this part of the subject a certain amount of repetition is, I fear, unavoidable.

The Increased Pressure and Increased Pressure-irritation Theories.

Von Graefe, who was the first to recognise and direct attention to the frequent occurrence of papillitis or "choked" disc (stauungs-papille, as he termed it) in cases of intracranial tumour, supposed that it was distinct from the descending optic neuritis which results from cerebral meningitis; and that it (the stauungspapille) was the result of increased intracranial pressure exerted on the cavernous sinus, with consequent obstruction of the venous return from the

orbit. This obstruction to the venous return was, he thought, intensified by the unyielding character of the sclerotic ring.

This theory was abandoned when it was shown that the communication between the superior ophthalmic and the anterior facial veins is so free that even complete obstruction of the cavernous sinus does not produce venous stasis and œdema in the optic papilla.

Modifications of the increased intracranial pressure theory were subsequently advanced, when it was shown that the subvaginal space surrounding the optic nerve is continuous with the subarachnoid space in the cranial cavity, and further (though the correctness of this supposed anatomical fact is denied by Leber) that coloured liquids injected into the cranial cavity not only find their way into the subvaginal space surrounding the nerve, but also (presumably through the lymph spaces in the optic nerve and lamina cribrosa) into the fundus oculi.

These modifications of the increased intracranial pressure theory suppose that, as a result of the increased intracranial pressure, which is so frequently present in cases of intracranial tumour, fluid is forced into the subvaginal sheath, which becomes over-distended; and that the papillitis is due either to (*a*) the venous stasis which results from compression of the optic nerve and its vessels by the fluid in the over-distended sheath, or (*b*) lymphatic engorgement. Schmidt believed that the neuritis was not the result of mere compression of the optic nerve, but that it was perhaps due to irritation, exercised by fluid forced into the lymph spaces.

In opposition to these modifications of the increased pressure theory, it has been argued (1) that in some cases of intracranial tumour, in which there is marked post-mortem evidence of increased intracranial pressure, there is no neuritis; (2) that in some cases of intracranial tumour in which there is no increased intracranial pressure there is neuritis; (3) that optic neuritis occurs in connection with small localised tumours as well as with voluminous new growths; (4) that in some of the cases in which there is neuritis there is no distention of the vaginal sheaths; (5) that neuritis is met with in conditions of cerebral atrophy, or rather with a wasted brain in which there are diffuse irritative, microscopical changes; (6) that in chronic hydrocephalus, in which there is great increase of the

intracranial pressure, neuritis is rarely seen; (7) that in cerebral hæmorrhage, with increased pressure, neuritis does not occur; (8) that in the rare cases of cerebral tumour in which neuritis is limited to one eye, it occurs in the eye on the opposite side to the brain lesion; (9) that the lymph spaces, which are supposed to pass from the sub-vaginal space into the optic nerve, do not exist (Leber); and (10) that experimentally produced increased pressure within the cranial cavity and vaginal sheath does not produce neuritis.

The last argument which Deutschmann has advanced, and which is based on an elaborate series of experiments, seems to me by far the most convincing.[1]

His experiments were made with the object of determining the following points. (I quote from the very full abstract of his work published in the *Ophthalmic Review*, April 1887.)

"(1.) What degree of hydrops of the optic nerve in animals is required to produce changes in the disc similar to those of the choked disc in man?

"(2.) Are there any conditions under which a moderate and even transient dropsy of the nerve, such as is found post mortem in man, is associated with the occurrence of choked disc?

"For the determination of the first question, he made injections directly into the nerve sheath. Dividing the superior rectus, he laid bare and cut through the optic nerve in front of the optic foramen; then drawing forward the distal end, and taking special care not to rupture the central vessels at their entrance into the nerve trunk, he injected warm sterilised agar-agar solution, the advantage of which is that at the body temperature it remains of a soft semi-fluid consistence, and is only very slowly absorbed. A ligature being then applied to the nerve, the divided muscle was sutured, and the wound closed; the whole with antiseptic precautions. Healing followed almost without trace of the operation. By filling the nerve sheath very forcibly, he obtained ophthalmoscopic evidence of a total arrest of circulation in the retina, and a few hours later a swelling and turbidity of the papilla much resembling the choked disc in man. Microscopic sections showed a well-marked compression of the nerve close to the globe, swelling and œdema of the papilla, and so forth, but no trace of

[1] *Ueber Neuritis Optica*, von Prof. R. Deutschmann, 1887.

true neuritis and perineuritis. When the injection was less forcible, however, though still sufficing to produce a more marked hydrops than is usually found in the human subject, he obtained only a temporary diminution of the arteries and an over-filling of the veins; moreover, these vascular changes disappeared in an hour or two, and microscopic examination several days later showed no trace of pathological change either in the nerve or the papilla.

"Deutschmann concludes that in animals a choking of the disc comparable with that which occurs in man can only be produced by pressure sufficient in amount to arrest the circulation. No one, he points out, has found a degree of hydrops in the human optic nerve approaching to this required amount; no one has found a condition of simple œdema without trace of commencing inflammation; no one has seen evidence of actual compression of the nerve; moreover, the frequent retention of good vision in such cases refutes the idea of an arrested circulation.

"In the next place, Deutschmann made injections into the cranial cavity in order to fill the nerve sheath by means of an excess of pressure within the skull. He used agar-agar, coloured with Indian ink, and rendered fluid by warmth. He repeated the injections from time to time in the same animal, and employed strictly antiseptic measures. He thus established not merely a transient excess of pressure in the skull, but one which was renewed from time to time; and by the antisepsis he obtained the effects of pressure without inflammatory complication. A well-marked injection of the optic nerve sheaths, fully equal in degree to that which is found in the human subject under conditions of morbid pressure in the cranium, and lasting for several weeks, was produced. Dissection showed that the sheath was forcibly distended, but that the papilla, the nerve trunk, and its sheaths were absolutely free from any trace of inflammation.

"From these experiments Deutschmann concludes that an excess of intracranial pressure, with distention of the optic nerve sheath, does not of itself suffice to produce the choked disc. The transient vascular changes visible after each rise of pressure cannot be regarded as the first stage of the morbid process in question."

It may perhaps be doubted whether the increased intracranial and intravaginal pressure, which Deutschmann produced in the manner

described above, is exactly comparable with, and could be expected to be attended with the same results as the increased pressure due to a growing, organised, living tissue material, such as a tumour. But be that as it may, these experiments, to my mind, afford by far the most striking evidence which has yet been advanced against the simple increased pressure view.

That increased intracranial pressure does play an important, though it would appear from Deutschmann's experiments a secondary, part in the production of papillitis in many cases of intracranial tumour, I am nevertheless still firmly of opinion.

So far as my experience enables me to judge, the signs of increased pressure are very generally found post mortem.

The absence of papillitis in some cases, in which there is well-marked evidence of increased pressure after death, can perhaps be explained by supposing that there is some mechanical impediment to the distention of the vaginal sheaths, possibly due to the pressure of the enlarged and swollen brain, or produced in some other manner; or, if there are lymph spaces passing from the vaginal space into the optic nerve and papilla, to the presence of some obstruction—such as thickening due to a former inflammation of the sheaths of the nerve—to the passage of the subvaginal and irritant-carrying fluid.

The fact that in cases of cerebral tumour (such, for example, as cerebral syphilis) a neuritis which has disappeared under treatment very rarely indeed returns, even although the cerebral lesion should take on fresh development, seems to lend support to this view, and it is to my mind an important argument against the "descending" neuritis theory.

Again, I agree with Broadbent in thinking, that the fact, that in those rare cases in which a cerebral tumour is associated with unilateral neuritis, the ocular affection has always been found on the opposite side to the brain lesion, instead of being in favour, as Stephen Mackenzie thinks, of a "descending" inflammatory process, can only be explained by supposing that there was some mechanical impediment to the entrance of fluid into the vaginal space, such as might be caused by the pressure of the swollen cerebral hemisphere in which the tumour is situated. The fact that the decussation in the chiasma is incomplete would, in my opinion, necessitate a "descending" in-

flammation being carried (granting, of course, that the inflammation was a true neuritis, descending along the nerve fibres) to *both* discs, rather than to the disc on the opposite side only. With reference to this point, and to the absence of papillitis in some cases of tumour in which there is increased intracranial pressure, the following statement, made by Dr. Broadbent at the debate on optic neuritis, is of importance:—"It should be remembered," he said, "that not only pressure but fluid was requisite, and in some cases, especially with large effusion into the ventricles, all fluid was squeezed out of the membranes, and the surface of the brain was dry and sticky. Again, the fluid might be prevented from entering the optic sheath, and he had been struck by two cases of cerebral tumour with hemiplegia, mentioned by Dr. Jackson, in which there had been unilateral optic neuritis in the eye of the paralysed side, the unaffected eye being on the same side as the tumour. He had had a similar case, which he had previously thought stood alone, and the tumour had apparently excluded the fluid from the optic sheath of that side."[1]

Again, I would ask, is it not the fact that in most of the cases of intracranial tumour in which optic neuritis is absent there is little or no evidence of increased pressure? My own observations would certainly lead me to suppose that this is so. And if it is a fact, it seems to me to be strongly in favour of the view that increased pressure does exercise an important influence in the production of papillitis. In the *Edinburgh Medical Journal* for January 1887, I have reported two cases of glioma with hæmorrhagic extravasations and the formation of large cysts. In the one, which was situated in the left frontal lobe, there was no increase of intracranial pressure and no neuritis; in the other, which was situated in the cerebellum, there was great increase of the intracranial pressure and intense neuritis. The tumour in the frontal lobe was eminently an irritative lesion, for it had, by a process of direct infection, extended across the anterior median fissure to the surface of the opposite hemisphere of the brain. In these two cases the only factor which could account for the production of neuritis in the one case, and its absence in the other, was increased pressure; for if an irritant produced by the tumour metabolism was

[1] *Transactions of the Ophthalmological Society of the United Kingdom*, vol. i., p. 110.

the only cause of optic neuritis, papillitis should have been present in the former of these cases, as it was in the latter.

It is because the Leber-Deutschmann theory recognises the importance of increased intracranial pressure, as well as the presence of an irritant, that I think it affords the most reasonable explanation in the majority of cases.

The fact that optic neuritis may be associated with small as well as with large tumours is probably, in some instances at all events, due to the circumstance, pointed out by Broadbent, that "intracranial pressure is not produced by the tumour, but by the effusion set up by it, and that this may be quite as abundant when the tumour is small as when it is large, while a large tumour may devour the brain substance as it grows."[1] The absence of neuritis in hydrocephalus is probably best explained by the absence of an irritant in the subarachnoid fluid, or perhaps in those cases in which the fontanelles are not closed, by the relief of pressure which is afforded by the yielding of the bones of the skull.

The fact that tumours which directly press upon the chiasma (and are therefore likely to press upon, flatten, and prevent distention of the vaginal sheaths) are in some instances attended with optic atrophy, *which is not preceded by papillitis* (*i.e.*, primary atrophy), is also perhaps an argument in favour of the increased pressure rather than of the descending neuritis theory.

In speaking of the distention of the nerve sheath, Gowers, who is an advocate of the descending neuritis rather than of the increased pressure theory, says:—"This condition is so frequently associated with optic neuritis, that it cannot be altogether without significance, although we may not be disposed to accept the theory of Schmidt-Rimpler, who sees in it the essential cause of the affection of the papilla. The facts that optic neuritis may occur without distention of the sheath, and that the latter may occur without the former, make it difficult to recognise a necessary casual relation between the two. Whatever the origin of the distention of the sheath, we must regard it, however, as exerting an influence on the neuritis, and it seems probable that under certain circumstances it may be the sole cause. Not long ago I made a post-mortem examination on a child,

[1] *Medical Ophthalmoscopy*, Second Edition, p. 110.

ten years of age, who was admitted into the University College Hospital with acute cerebral symptoms, the exact nature of which was obscure. An ophthalmoscopic examination, however, showed intense double optic neuritis, with great swelling, large hæmorrhages, and extreme distention of the veins. There were also several spots of choroidal damage, characteristic of inherited syphilis. In a few days the child died; we found an extensive cerebral hæmorrhage, and many of the arteries presented well-marked syphilitic disease. There was no lesion, however, which could be regarded as a cause of the optic neuritis. But the orbital plates of the frontal bone were thickened to five or six times the normal, and the optic foramina were much narrowed. The sheaths of the optic nerves were enormously distended, and to the narrowing of the foramina and distention of the sheaths the optic neuritis was apparently due." And again he states, "In all cases in which distention of the optic sheath occurs it probably intensifies the inflammation, however arising."[1]

The facts that neuritis occurs in some cases of intracranial tumour in which there is no increased intracranial pressure, and no distention of the sheath, and indeed in some cases of cerebral atrophy with diffuse irritative changes, only show that optic neuritis may be produced by other causes than increased pressure—a point which I fully allow. These facts do not prove that increased pressure is of no importance in those cases of intracranial tumour—and they are the majority—in which it does occur.

Again, as Broadbent argues, in cerebral hæmorrhage there is not the persistent high pressure that there is in most cases of tumour; and to this I would add that in those cases in which the cerebral hæmorrhage is so large as to give rise to great increase of the intracranial pressure, the case is usually fatal, and there is therefore no time for optic neuritis to be developed.

Again, in some rare cases of hæmorrhagic extravasation at the base of the brain, in which presumably blood makes its way into and distends the vaginal sheaths, optic neuritis may be developed. This is certainly very rare. I have reported a case of this kind, in which intense neuritis, rapidly followed by total loss of vision, was produced at such an early stage as to suggest that the extravasated

[1] *Medical Ophthalmoscopy*, Second Edition, pp. 106 and 108.

blood had made its way from the base of the brain into the vaginal sheaths, and had interfered with the functions of the optic nerve and

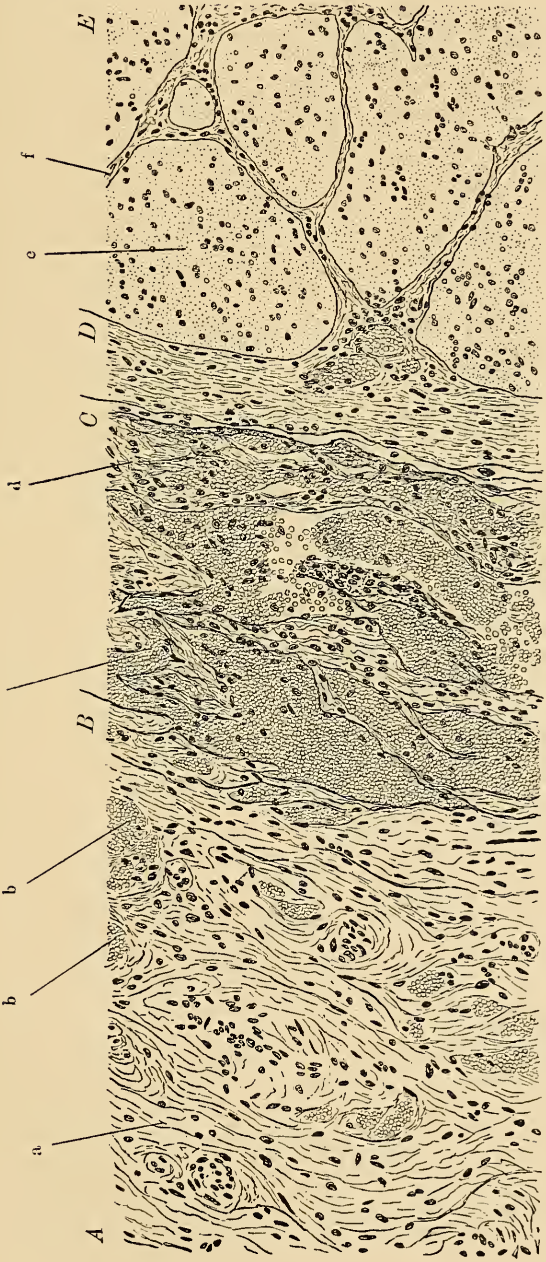

Fig. 13.—Camera lucida drawing of a transverse section through the optic nerve and its coverings in a case of basilar meningeal hæmorrhage, due to the rupture of an aneurism at the base of the brain, showing the subvaginal space distended with blood, inflammatory changes in the fibrous covering of the nerve, and an excess of corpuscular elements in the nerve trunk itself. (From a preparation stained with borax-carmine, cleared with alcohol and oil of cloves, and mounted in xylol balsam. Hartnack, oc. 3, obj. 8; tube out; and drawing reduced from $11\frac{1}{4}$ to $6\frac{1}{2}$ inches).

A to *B*, a portion of the outer or dural sheath; *B* to *C*, the subvaginal space; *C* to *D*, the pial sheath of the nerve; *D* to *E*, a portion of the nerve trunk.

The letter *a* points to the fibrous tissue of the dural sheath, which is studded with numerous large, deeply-staining connective tissue corpuscles; *b b*, to collections of red blood corpuscles between the bundles of fibres; *c*, to masses of red blood corpuscles which fill the subvaginal space; *d*, to the trabeculæ of connective tissue passing across the subvaginal space; *e*, to a bundle of nerve fibres placed between the pial sheath (*C* to *D*) and the fibrous tissue trabecula of the nerve (*f*).

The corpuscular elements, both in the nerve itself and in the trabeculæ of the subvaginal space, are in excess. The case is reported in the *Edinburgh Medical Journal* for August 1886.

produced the papillitis, partly by direct pressure and partly by exciting inflammatory changes in its immediate neighbourhood. That the papillitis might have been produced in this case by a "descending" neuritis, must of course be allowed; but it is, I think, more difficult to account in this way, than in the manner which has been suggested above, for the fact that vision was so completely lost at such an early stage of the case, and for the early occurrence of such intense papillitis.

That blood which is extravasated at the base of the brain may find its way into the vaginal sheaths, and may produce inflammatory changes, is shown by another case (rupture of an aneurism at the base of the brain) which I have reported in the same journal,[1] and a microscopic section of which is represented in Fig. 13.

The important influence which some forms of pressure have on the production of optic neuritis is also shown by "a case of Mr. Lawson's" (quoted by Hilton Fagge), "in which the presence of a hydatid cyst within the orbit caused an extreme state of "choked disc;" four days after puncture of the tumour the engorgement was found to have almost entirely disappeared.[2] The same rapid subsidence of the optic atrophy may also follow the evacuation of a cerebral abscess.

For all these reasons, then, I am disposed to think that increased intracranial pressure is in many cases an important factor in the production of the double optic neuritis in cases of intracranial tumour; though I am not prepared to assert that *per se* it is the primary or the sole and sufficient cause of the condition.

The Vaso-motor Theory.

Dr. Hughlings Jackson lends the great weight of his authority to this theory, which supposes that the tumour induces changes in the grey matter of the brain in its neighbourhood, and that these alterations in the cerebral grey matter, cause, through the vaso-motor nerves, vascular changes in the optic discs, which result in inflammation — in other words, in optic neuritis. In the debate at the Ophthalmological Society Dr. Hughlings Jackson said—and, so far as I know, he still adheres to the opinion which he then expressed—

[1] *Edinburgh Medical Journal*, August 1886, p. 97.
[2] *Principles and Practice of Medicine*, vol. i., p. 520.

"The hypothesis which *seems to me most plausible* when the whole of the non-localising symptoms, and such conditions of the centres as epileptiform seizures imply, are taken into account, is that optic neuritis results doubly indirectly from vaso-motor action. I think," he said, "that optic neuritis may be a doubly indirect result of local gross organic disease: that, first, there are changes of instability about the tumour; that next these lead on to discharges, by intermediation of vaso-motor nerves, to repeated contractions, with subsequent paralysis of vessels of the optic nerves or centres, and then, at length, to that trouble of nutrition which is optic neuritis. I repeat that such induced changes are causes of spasm of big muscles. Moreover, in cases of epilepsy proper, we see often enough effects produced by discharges in arteries and in viscera, as well as convulsions by discharges in big muscles."

The following facts and arguments seem to negative the vaso-motor theory:—

(1.) As Gowers, Leber, and others have argued, it is doubtful if vaso-motor alterations can or do produce true inflammatory changes.

(2.) The vaso-motor nerves, which are supposed to be concerned in the production of optic neuritis in the manner described above, and which (since tumours in all parts of the cranial cavity are attended with neuritis) must be distributed to all parts of the cerebral tissue, have never been demonstrated.

(3.) Other irritations of the cerebral tissue, such as those which are produced in the neighbourhood of hæmorrhagic extravasations, patches of softening, &c., do not produce optic neuritis.

(4.) If optic neuritis can be produced in this way (*i.e.*, by vaso-motor irritation), inflammatory changes ought also to be produced in the internal ear and in the areas of distribution of the other cerebral nerves. (This point will be more fully discussed in connection with the descending neuritis theory.)

The Descending Neuritis Theory.

This view, which supposes that inflammatory changes make their way downwards from the cranial cavity to the optic nerve and optic papilla, either (1.) through the membranes, blood-vessels, and connective tissue, or (2.) through the optic nerve itself, has much to be said

in its favour. That optic neuritis may be, and frequently is, produced in this manner, every one will, I presume, allow; and that optic neuritis associated with some intracranial tumours is produced by such a descending inflammation I am fully prepared to admit; but I am not convinced that this is its usual or most common cause.

Many of the arguments which have been previously advanced in favour of the view that increased pressure is an important factor in the production of papillitis are more or less opposed to the descending neuritis view.

All these arguments need not be again repeated, but the following are some of the chief :—

(1.) In many cases of intracranial tumour in which well-marked papillitis was present during life, there is absolutely no appearance of meningitis after death.

This statement applies to the naked-eye appearances. Whether under such circumstances inflammatory changes can be demonstrated by means of the microscope I am not prepared to say; but in some instances in which I have examined the condition of the membranes at the base, and indeed in other parts of the cerebrum, I have failed to satisfy myself that such inflammatory changes were present.

(2.) Deutschmann argues that it is doubtful if the inflammatory changes which have been found in the optic nerve are "descending" or "ascending" in character; he indeed seems to suppose that, in many instances at all events, the inflammation commences at the back of the eyeball and ascends towards the cerebrum.

(3.) If it is the fact that in those cases of intracranial tumour in which there is no optic neuritis, increased intracranial pressure is more frequently absent than in the average run of cases—and my own observation would lead me to suppose that such is the case—this circumstance seems to me a strong argument against the descending neuritis view.

(4.) The fact that optic neuritis, which has once subsided, rarely relapses and returns, even although the intracranial tumour with which it is associated takes on renewed activity, seems also opposed to the descending neuritis theory; indeed, judging from analogy, I should have supposed that, if the condition is a true neuritis, one attack would predispose to, rather than prevent, the production of a second.

(5.) The fact that in unilateral papillitis associated with tumour, the papillitis is usually (?always) on the opposite side to the tumour, is also, to my mind, a strong argument against the descending inflammatory theory.

(6.) The fact that tumours in the region of the pituitary body, which one would have thought would be specially liable to excite inflammatory changes in their immediate neighbourhood, produce, in some instances at all events, primary optic atrophy, and not optic neuritis, is also perhaps an argument against the descending neuritis view.

(7.) The fact that deafness very rarely results from intracranial tumour, while complete blindness from papillitis is common, is also, I think, opposed to the descending neuritis theory; for if the papillitis is due to a "descending inflammatory" process, why should the auditory nerve not be affected with "descending inflammation" too? It may of course be said that the inflammation does descend along the auditory nerve, and that we are unable to recognise it; while we are able to see the papillitis by means of the ophthalmoscope. The argument is, however, fallacious; for, although the minor degrees of inflammation of the auditory nerve might for this reason pass unrecognised, the more severe inflammatory changes ought to produce deafness, just as the more severe forms of papillitis do produce blindness. I see no reason why severe inflammatory changes in the auditory nerve terminations, and delicate end-organ of hearing, should not produce deafness, just as we know severe inflammatory changes in the optic disc and end-organ of sight produce blindness. And since deafness does not, except in the very rarest instances, result from intracranial tumours, I conclude that a severe form of descending neuritis in the auditory nerve does not occur. This constitutes, in my opinion, an argument against the view that the papillitis is produced by a (to be strictly accurate and logical) *severe* inflammatory change descending *along the nerve fibres.*

In this connection, it is important to remember the suggestion thrown out by Gowers, that "a very slight degree of descending inflammation may be the connection between a neuritis of the cerebral and one of the ocular extremities of the nerve. We know," he says, "from the occurrence of neuritis in general diseases, that the

optic papilla is a structure in which inflammation readily occurs, and it seems as if a slight process of irritation descending the nerve was sufficient to light up a considerable papillitis. We know also that a cerebral tumour is the seat of irritative changes, and it seems probable that in some cases such irritation may be widely spread, and may reach and descend the optic system of fibres."[1] Dr. Buzzard fully realises the importance of the argument, that if the papillitis is the result of a descending inflammation, or of vaso-motor changes induced by the irritation of cerebral grey matter in the neighbourhood of the tumour, the inflammation ought not to be limited to the optic, but should also involve the other cerebral nerves. His remarks on the vaso-motor theory at the debate of the Ophthalmological Society, were as follows:—" But to make this explanation feasible, it appeared to him that it was necessary either to account for the optic nerve being singled out as the solitary subject of the pathological change, or to show that it was not singled out, but that an analogous condition was apt to prevail in the course of the cranial nerves. The latter course, perhaps, offered most promise. He was disposed to think that in many cases the pains in the head, associated with intracranial tumour, were due to neuritis of the fifth nerve, for although, perhaps, they were as a rule not distinctly localised, yet the patient would sometimes refer them very clearly to the district of that nerve, and tender points would occasionally be found in the situations similarly affected in trigeminal tic. It might be a question, too, he thought, whether the vertigo, which was of not uncommon occurrence in cases of intracranial tumour, might not be due to changes in the nerves supplying the external ocular muscles, producing waves of disordered function corresponding to those which Dr. Jackson had described as causing temporary loss of sight in the case of optic neuritis. Was it possible, again, that the vomiting and slowing of the pulse might represent an affection of the pneumogastric brought about by the same cause as that which produced optic neuritis, and that the sudden or rapid death, which Dr. Jackson had mentioned as one of the contingencies of optic neuritis from intracranial disease, might also be explained by a more severe influence exerted upon the nerve?"[2]

[1] *Transactions of the Ophthalmological Society of the United Kingdom*, vol. i., p. 107.
[2] *Ibid.*, vol. i., p. 98.

These, then, are the chief reasons which lead me to doubt whether a descending inflammatory process (more especially an inflammation descending along the nerve fibres) is the usual cause of the papillitis associated with intracranial tumours.

To sum up, it appears to me that the double optic neuritis associated with intracranial tumours is not always produced in the same manner. It must be allowed that in some cases it is due to a "descending" inflammation, but this does not appear to me to be the usual or most common cause of the condition. The pressure-irritation theory of Leber and Deutschmann is, in my opinion, the most likely explanation in the majority of cases. Increased intracranial pressure is an important, though, as Deutschmann suggests, a secondary and favouring cause of the condition. The essential and primary cause is probably in most cases, as Leber and Deutschmann have suggested, the presence of an irritant in the cerebro-spinal fluid. Increased intracranial pressure is an important factor, inasmuch as it forces the arachnoid fluid into the subvaginal space, where the irritant contained in the subarachnoid fluid produces inflammatory results in the vaginal space, optic nerve, and optic papilla. But before the Leber-Deutschmann theory can be definitely adopted, the presence of an irritant, and its exact nature, require, I think, further demonstration. The weight of evidence seems to me strongly opposed to the vaso-motor view.

CHAPTER IV.

MOTOR DERANGEMENTS—PARALYSIS—SPASM—TREMOR—CONTRACTURE.

Paralysis.

Derangements of motion, more especially paralysis and spasm, are of frequent occurrence in cases of intracranial tumour, and are of great importance from a localising point of view. The extent and distribution of the paralysis is of course very different in different cases, for a tumour may involve any part of the intracranial neuro-motor apparatus :—(*a*) cortical centres ; (*b*) conducting fibres, in the centrum ovale, internal capsule, crus cerebri, pons Varolii, or medulla oblongata ; or (*c*) intracranial portions of the (peripheral) cranial nerves.

Now, since both at the top and bottom of the neuro-motor apparatus there is anatomical separation as well as physiological differentiation, it follows that tumours, which involve the cortical centres or peripheral nerves, are much more likely to produce localised paralyses than tumours which involve the great bands of conducting fibres (see Fig. 14).

Cortical or subcortical tumours of limited extent may produce very limited and localised paralysis—paralysis of one limb (monoplegia), or of part of a limb—just as excision of limited portions of the cortex produces very localised (though temporary) paralysis in the lower animals. As the tumour increases in size, adjacent motor centres may become involved, and the lesion may gradually extend, until ultimately all the motor centres, or all the conducting fibres proceeding from them, are involved, and the paralysis assumes the ordinary hemiplegic type (*i.e.*, involves the face, arm, and leg on the opposite side of the body).

Cortical or subcortical tumours, which are situated in the middle line and which involve the motor area in each hemisphere, may

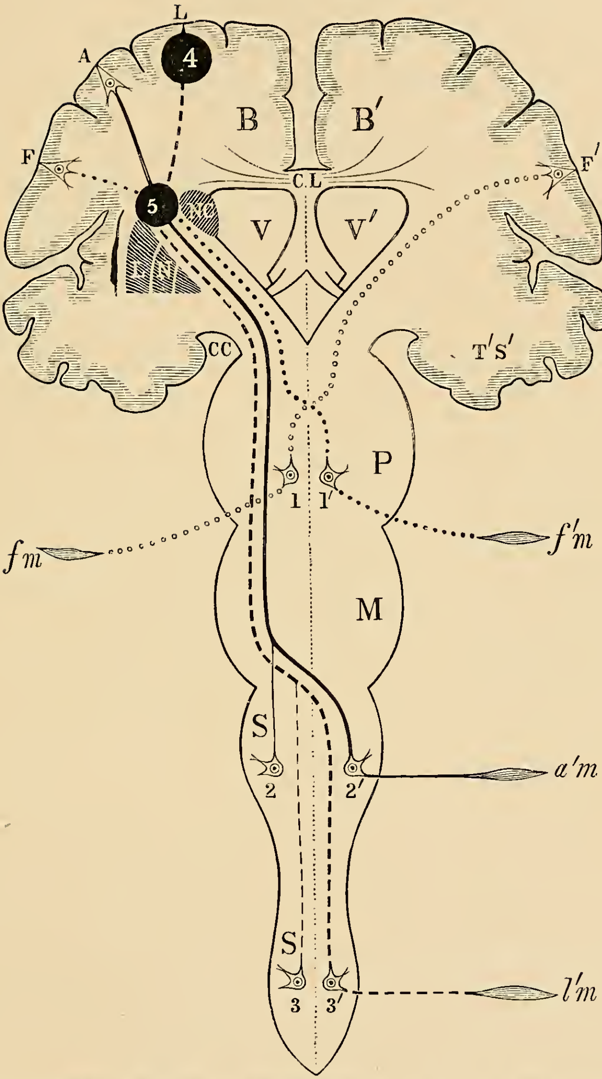

FIG. 14.—Diagram showing the arrangement of the motor tracts for both sides of the face, the left arm, and the left leg; and the distribution of the paralysis produced by lesions in different parts of the cerebrum.

B, the right, and *B′*, the left cerebral hemisphere; *P*, the pons Varolii; *M*, the medulla oblongata; *S*, *S*, the spinal cord; *V′ V′*, the lateral ventricles; *NC*, the nucleus caudatus; *L N*, the lenticular nucleus; *C C*, the crus cerebri; *T′ S′*, left temporo-sphenoidal lobe; *F*, *A*, *L*, cortical centres for the face, arm, and leg in the right hemisphere; *F′*, cortical centre for facial muscles in the left cerebral hemisphere; 1, 1′, right and left facial nerve nuclei; 2, 2′, right and left nerve nuclei for the upper extremity; 3, 3′, right and left nerve nuclei for the lower extremity; *fm* and *f′m*, right and left facial muscles; *a′m*, muscles of left upper, and *l′m*, muscles of left lower limb; 4, localised lesion in the cortex, producing paralysis of the opposite leg; 5, lesion of the pyramidal tract as it enters the internal capsule, producing hemiplegia on the opposite side of the body (paralysis of the face, arm, and leg).

produce limited paralysis on both sides of the body. Paralyses of this kind are certainly very rare, but it is quite conceivable that a tumour of the vertex might involve both leg centres and so produce paraplegia.

Tumours involving the corpus callosum may extend into each centrum ovale, invade each internal capsule, and so produce paralysis (more or less complete hemiplegia) on both sides of the body. Further, tumours which involve or press upon the motor tract in the pons Varolii or medulla oblongata, not infrequently produce bilaterally distributed paralysis, which often involves, but seldom completely paralyses, all four extremities.

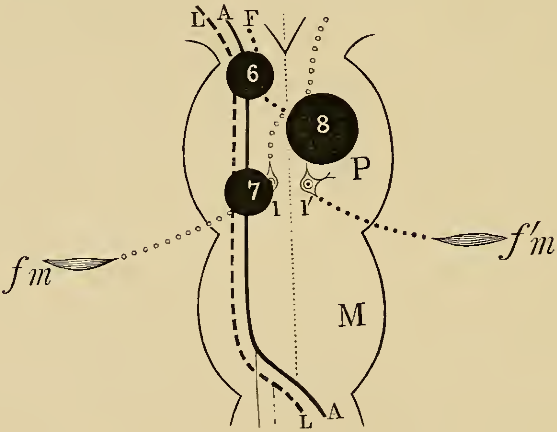

FIG. 15.—Diagram showing the arrangement of the motor tracts for both sides of the face, the left arm, and the left leg in the pons Varolii, and the distribution of the paralysis produced by lesions of different parts of it.

6, lesion in the upper part of the right side of the pons, producing paralysis of the face, arm, and leg on the opposite (left) side; 7, lesion in the lower part of the right side of the pons Varolii, producing paralysis of the face on the same, and of the arm and leg on the opposite side; 8, lesion in the middle of the left side of the pons Varolii, producing paralysis of both sides of the face, and of the arm and leg on the opposite side,—the conducting tracts passing to the right arm and leg are not shown in the left side of the pons, but they would obviously be interrupted by this lesion.

It must be remembered that in many cases of cerebral tumour there is more than one focus of new growths; and that it is quite possible to have irregular forms of paralysis where there are multiple lesions.

Tumours which involve one pyramidal tract, either in the internal capsule, crus cerebri, pons Varolii, or medulla oblongata, usually cause hemiplegia, for they usually involve, more or less completely, all the bundles of fibres of which the pyramidal tract is composed.

When the lesion is above the crus cerebri, the hemiplegia is of the ordinary type. When the lesion involves the crus cerebri, or is situated in the pons Varolii or medulla oblongata, one or other of the irregular or alternate forms of hemiplegia may result, viz. :—(1.) Paralysis of the face, arm, and leg on the opposite side to the lesion, and of the muscles supplied by the third nerve on the same side; (2.) Paralysis of the arm and leg on the opposite, and of the face on the same side as the lesion (see Fig. 15); (3.) Paralysis of the arm and leg on the opposite, and of the muscles supplied by the facial (seventh) and sixth nerve (external rectus) on the same side; (4.) Paralysis of the arm and leg on the opposite, and of the face on both sides (see Fig. 15); (5.) Paralysis of the face, arm, and leg on the opposite side, and anæsthesia (from paralysis of the fifth nerve) on the same side of the face; (6.) Paralysis of the arm and leg on the opposite, and of the tongue on the same side.

Tumours at the base of the brain may involve one or more of the peripheral nerves (third, fourth, sixth, seventh, &c.) on one or both sides, and produce a localised peripheral paralysis, or different combinations of peripheral paralysis. In cases of this kind the distribution of the paralysis is different from that due to a localised cortical lesion, and the condition of the paralysed muscles is usually characteristic.

Further, in addition to involving one or more of the peripheral nerves, a tumour at the base may obstruct the middle cerebral artery, or some of its branches, and may consequently produce softening in the motor area of the cortex (on the same), and therefore hemiplegia on the opposite side of the body. The form of alternate hemiplegia in which the face, arm, and leg are paralysed on the opposite, and the muscles supplied by the third nerve on the same side as the lesion, and which, when sudden and acute, is distinctive of a lesion of the crus cerebri, may therefore be produced by a tumour, usually a syphilitic gumma, at the base of the brain involving the trunk of the third nerve and the trunk of the middle cerebral artery.

The condition of the paralysed muscles (their state of nutrition, and their electrical reactions), the condition of the reflexes, and the presence of twitchings, tremors, and spasms, depend upon the position and character of the lesion. Whether the resulting paralysis is central or peripheral—or, more correctly, whether it is due to a lesion (1)

above or (2) *in* or *below* the nerve nuclei of the affected muscles—is of course the most important point.

It is important to note, that in the various forms of alternate hemiplegia the muscles which are paralysed on the same side as the lesion present the atrophy, electrical reactions, and reflex alterations which are characteristic of peripheral paralysis, whereas those on the opposite side present all the characteristics of a paralysis due to a central (*cerebral*) lesion (*i.e.*, a lesion above the nerve nucleus).

Localised convulsive twitchings, or attacks of "Jacksonian" epilepsy, often precede or are associated with the paralyses which are due to cortical or subcortical lesions; for the process of irritation often precedes, or is associated with, the process of destruction which is produced by a tumour.

A rhythmical tremor, occurring on attempts at voluntary movement, which closely resembles the tremor of cerebro-spinal sclerosis, and which is due to pressure upon, and interrupted conduction through, the fibres of the pyramidal tract, is met with in some cases. It is not infrequently due to the pressure of a cerebellar tumour upon the pons Varolii or medulla oblongata; and I have met with cases in which it had apparently resulted from the pressure of new growths in the region of the centrum ovale, upon the motor fibres of the internal capsule.[1]

In this connection I may say that I am doubtful if *simple* pressure on the fibres of the pyramidal tract (*i.e.*, pressure without irritation) produces exaggeration of the knee jerk, as is generally supposed.[2]

It will be apparent from what has just been stated, that the extent and distribution of the paralysis, and the condition of the paralysed muscles, reflexes, &c., afford in many cases most important information as to the exact position of the tumour.

In all cases of intracranial tumour in which spasm or paralysis appears to be due to a cortical lesion, it is advisable to refer to the experimental results obtained by Ferrier and others in the lower animals. It is perhaps premature to conclude that the results of cortical irritation in the monkey exactly correspond with those in man; but the

[1] See a case which I have reported in the *Edinburgh Medical Journal* for January 1887, p. 623.

[2] *Ibid.*

effects of disease in the human subject seem to show that the plan of arrangement in the two brains is, as regards the motor centres at all events, very similar.

In Figs. 16 and 17 the position of the motor centres, as determined by Ferrier on the lateral and upper aspects, and in Fig. 18 by

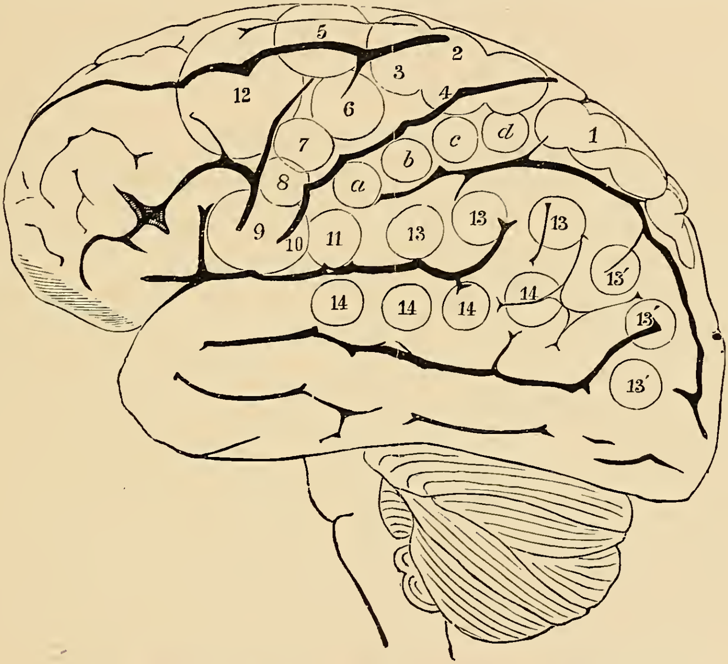

Fig. 16.—Lateral view of the human brain, showing the position of the motor centres. (After Ferrier.)

1. Centre for opposite leg and foot.
2, 3, 4. Centres for movements of arms and legs, such as are concerned in climbing, swimming, &c.
5. Centre for extension forwards of the arm and hand.
6. Centre for the supination of the hand and flexion of the fore-arm.
7, 8. Centres for elevators and depressors of mouth respectively.
9, 10. Centre for the movements of the lips and tongue in articulation.
11. Centre for the platysma ; retraction of the angle of the mouth.
12. Centre for lateral movements of the head and eyes, with elevation of the eyelids and dilatation of the pupil.
a, *b*, *c*, *d*. Centre for the movements of the hand and wrist.

Centre 5 involves the posterior end of the first ; 12, the posterior end of the second ; and 9, the posterior end of the third frontal convolutions.

Centres 2, 3, 4, 6, 7, 8, and 9 are placed on the ascending frontal convolution.

Centre 1 involves the posterior parietal lobule, and centres 11, *a*, *b*, *c*, and *d*, are placed on the ascending parietal convolution.

Centres 13 and 13′ (Ferrier's centres for vision) are placed on the supramarginal convolution, angular gyrus, and occipital lobe.

Centres 14, 14 (Ferrier's centres for hearing) on the superior temporo-sphenoidal convolution.

Victor Horsley and Schäfer on the inner aspect of the brain of the monkey, is shown.

Stated in detail, the results obtained by Horsley and Schäfer were as follows :—

"The general results of our experiments," they say, "will best be understood by a reference to the accompanying figure (Fig. 18). Thus, in the part of the marginal convolution marked 1, 1′, extending from

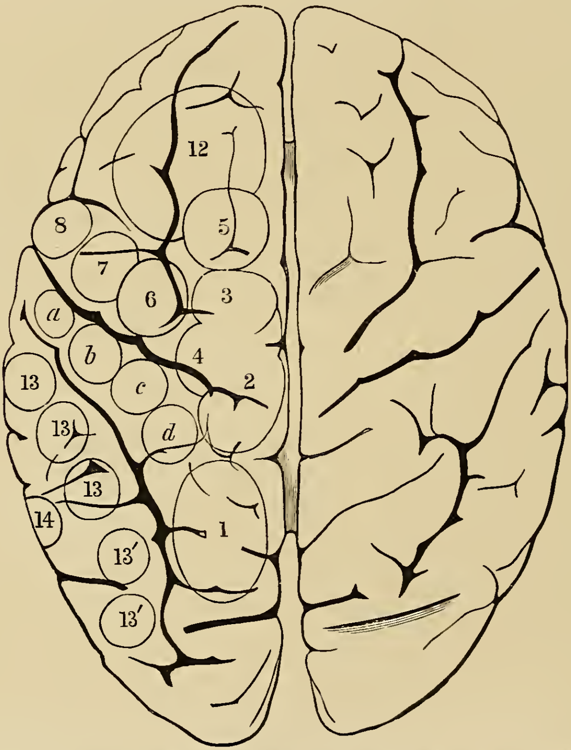

Fig. 17.—Upper view of the human brain, showing the position of the motor centres. (After Ferrier.)

just in front of the small vertical sulcus y to a point on a level with the anterior third of the small antero-posterior sulcus x, excitation is followed by either movements of the fore-arm (flexion or extension), or by adduction of the arm and retraction of the shoulder combined with outward rotation, or by any of these movements of shoulder and arm either combined or succeeding one another in definite order, according to the point in the area which is stimulated. Retraction

of the shoulder (combined with flexion of the fore-arm) is alone produced by excitation of the posterior portion of this area, and when manifested as the result of excitation applied here, is apt to be associated with movements of the trunk, pelvis, or hip, which, as the overlapping of the contours of the areas shows, may also be called forth by excitation of this part.

" In the next area, 2, 2′, we get movements of the trunk muscles as the result of excitation, the chief effect produced being a rotation of

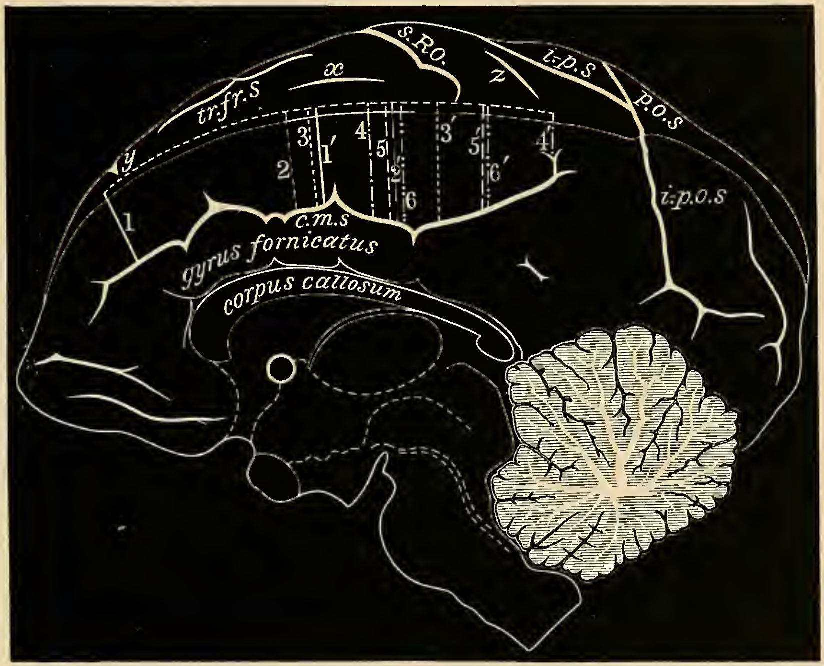

FIG. 18.—Inner aspect of the right hemisphere of the brain of a monkey, showing the position of the motor areas in the marginal convolution, according to Horsley and Schäfer.

The centres overlap one another.

From 1 to 1′ = centre for the upper extremity.
2 to 2′ = centre for the trunk muscles.
4 to 4′ = centre for the lower extremity.

The movements from before backwards seem to be arranged in the following order :—(1.) movements of the head ; (2.) of the fore-arm and hand ; (3.) of the arm at the shoulder ; (4.) of the upper (dorsal) part of the trunk ; (5.) of the lower (pelvic) part of the trunk ; (6.) of the leg at the hip ; (7.) of the lower leg at the knee ; (8.) of the foot and toes.

the body to the opposite side to that stimulated, combined with an arching of the spine, with the concavity directed towards the opposite

side. In the anterior part of the area the chief effect is upon the dorsal region, but in the posterior part it is upon the lumbar region and pelvis.

"This area is largely overlapped by the next one, 3, 3′, excitation within which is followed by movements of the hip, at some points the flexors only, at others the extensors only, at others both sets of muscles being called into contraction simultaneously. As will appear from the overlapping of the areas in the figure, these movements of the hip are apt to be associated with the rotatory and bending movements of the trunk above mentioned; but in the anterior part of the area it is generally the rotation of the trunk and pelvis which is first seen, and this is followed by hip movements, whereas in the centre of the area, movements of the hip may be the first to appear, or with a very weak excitation may be the only ones visible.

"The next area, 4, 4′, is very extensive. It considerably overlaps the areas 2, 2′, and 3, 3′, and extends to the posterior limit of the convolution. Its excitation calls up contractions of the thigh muscles, and especially of the hamstrings, which in some parts are the only muscles affected by weak stimulation—indeed, in some instances the contractions of the individual hamstring muscles were perfectly localised. But in most parts of the area, as the overlapping of the contours show, these movements are associated with those of other muscles, viz., anteriorly with the trunk and hip muscles, and posteriorly with muscles which move the ankle and toes. These associated movements may be simultaneous, but are most commonly successive, as when by stimulation of one point there was produced, first, a contraction of one of the abdominal muscles, then of one of the thigh muscles, and then of one of the muscles which move the ankle.

"In like manner the area marked 5, 5′, may be looked upon as the specialised part from which the movements of the ankle are controlled, these being usually the first to appear on exciting the area, although very generally associated with or followed by movements of the hip and knee. And 6, 6′, may for a similar reason be looked upon as specially controlling certain movements of the toes, generally associated, however, with other movements of the lower limb. As before mentioned with regard to the other areas, the particular move-

ments called forth differ according to the point in the area which is excited, but our experiments do not as yet enable us to make sufficiently positive assertions as to the localisation of these specialised points."[1]

See also, with reference to the more exact position of the motor centres, Victor Horsley's paper on the "Topography of the Cerebral Cortex," in the *International Medical Journal* for April 1887, page 342; and Horsley and Schäfer, "A Record of Experiments upon the Functions of the Cerebral Cortex,"—*Philosophical Transactions*, vol. 179 (1888), B., pp. 1-45.

The causes of paralysis in cases of cerebral tumour are numerous.—(1.) In some cases the paralysis is due to *destruction* of some part of the intracranial neuro-motor apparatus (centres, conductors, peripheral cranial nerves); and, as has been previously stated, this destruction may either be the *direct* result of the lesion, or due to obstruction of the blood supply—*i.e.*, *indirect*.

It must be remembered that slow and gradual destruction of a large portion of the motor cortex (*i.e.*, motor grey matter) is not necessarily attended with paralysis (see Figs. 1, 2, 3, and 4, pp. 11-14); but that in those cases of cerebral tumour in which the paralysis is due to this cause, the loss of motor power is usually permanent.

(2.) In others, the paralysis is due to the *pressure* of the tumour, or of the inflammatory products which surround it, upon conducting (motor) fibres. In cases of this kind, the extent and degree of the paralysis may vary from time to time in consequence of variations in the degree and severity of the pressure.[2]

(3.) In other cases the paralysis is temporary, and due to passing (? functional) changes in the motor centres. Post-epileptic paralysis, which follows the spasms of "Jacksonian" epilepsy (and which is probably due to exhaustion of the grey matter which was violently discharged during the attack), is the best known form of functional cerebral paralysis.

Another variety, which is neither preceded by spasm nor unconsciousness, and in which there is sudden but temporary failure of motor power, is much more rare. I have, however, met with several well-

[1] *Proceedings of the Royal Society of London*, vol. xxxvi., p. 439.

[2] See a case reported by me in the *Edinburgh Medical Journal* for Jan. 1887, p. 623.

marked instances, and am disposed to think that it is due either to temporary alterations (arrest) in the blood supply, or to inhibition of motor grey matter.[1]

Spasms and Convulsions.

The increased and perverted function, which is so frequently produced in motor nerve tissue by intracranial tumours, is manifested externally in the form of spasms and convulsions, and the lesion is technically termed a "discharging" one. It is obvious, as Dr. Hughlings Jackson, who introduced the terms "discharging" and "destroying," has pointed out, that it is not the tumour itself which discharges, but the motor nerve tissue which is irritated by it.

Theoretically a tumour or other "coarse" lesion may cause discharge of motor grey matter, either by *directly* irritating it; or *indirectly* (1) by interfering with the nutrient supply (pressing upon and obstructing the blood-flow through its nutrient vessels, or irritating their vaso-motor nerves); or (2) by reflexly irritating it, the irritation passing, perhaps, in some instances, in the first place to the convulsive centre in the medulla.

Further, Duret has shown that spasms and convulsions may be produced by irritating sensory nerves in the dura mater; but the spasms which are produced in this way have not the well-defined and characteristic features of the localised epileptiform convulsions, due to irritation of the motor cortical centres, which will presently be described.

The character of the convulsions, whether tonic or clonic, depends, in part at least, upon the composition of the nerve tissue which is irritated (whether grey or white matter).

Clonic spasms and epileptiform convulsions, which are by far the most important forms of spasms met with in cases of intracranial tumour, may theoretically result from irritation of motor grey matter either in the cerebrum itself, in the pons Varolii, or medulla oblongata; but are, I think, in the great majority of cases due to discharge of motor grey matter (motor centres) in the cerebral cortex.

[1] See a case which I have reported in the *Edinburgh Medical Journal* for Dec. 1878, p. 501, in which the form of paralysis was a striking symptom.

The extent and distribution of the spasms are very variable. In some cases a single muscle or group of muscles is involved; in others the muscles of one limb are affected; in others, again, the spasm is unilateral, while in some the spasm is bilateral, and affects all, or almost all, the muscles of the body.

In localised epileptiform convulsions, or attacks of Jacksonian epilepsy, as they are commonly termed, there is usually no loss of consciousness; but in those cases in which the spasm is from the first, or in which it subsequently becomes, general and bilateral, consciousness is usually, but not invariably, lost, and the patient foams at the mouth; in short, the attack exactly resembles an ordinary (idiopathic) epileptic fit. In the *Edinburgh Medical Journal* for January 1879, p. 599, I have described a case of this kind, in which there was no loss of consciousness, although the spasms were bilateral and very severe. This is extremely rare.

Another characteristic feature of the localised convulsions of Jacksonian epilepsy is the fact that they are usually followed by temporary localised paralysis, affecting those muscles which were first or most convulsed. Dr. Hughlings Jackson thinks that after the bilateral spasms of ordinary epilepsy there is also some paralysis, but that it is less perceptible, being universally or thinly spread out over the whole muscular system.

Now it is important to remember that the discharge, which results from the irritation which a tumour or other "coarse" lesion produces in a limited portion of the motor area of the cerebral cortex, has the same strong tendency to extend to, and to involve, adjacent and more distant centres, and so eventually to produce general and bilateral convulsions, as the discharge induced by electrical irritation of a localised portion of the motor cortex in the lower animals. In both cases the spread or march of the spasm is not haphazard, but follows a definite course. The following case is a striking illustration of many of the points which have just been mentioned.

M. C., aged thirty-seven, was admitted to the Newcastle-on-Tyne Infirmary under my care on May the 23d, 1875, suffering from right-sided convulsions and hemiplegia.

Seven years previously she had received a severe blow on the left side of the head, which had fractured her skull. After the immediate

effects of this injury were recovered from, she had enjoyed good health, complaining occasionally of a feeling of "pins and needles" in the right forefinger and thumb. The present attack commenced on May 10th, after a heavy drinking bout.

On examination, a depression, the size of a threepenny piece, could be felt two inches above the left ear; the right arm and leg were completely paralysed; and every few minutes the patient was seized with a convulsion.

The fits were of three kinds, and may, for convenience of description, be termed *slight*, *moderate*, and *general*.

In the *first* or *slight* form the muscles of the face and neck were alone affected. Both eyes were firmly closed, and the right corner of the mouth drawn down in tonic spasm, the platysma being rigid. The eyes were then partly opened; the head and eyeballs slowly rotated to the right. Clonic spasms next occurred in both eyelids, the right being affected much more powerfully than the left; and in the muscles of the tongue and right side of the face and neck, the platysma being chiefly affected. After a short interval, the spasms became less frequent, the head and eyeballs were slowly turned back to the middle line, the eyelids were widely dilated, and the patient presented an animated appearance. The eyeballs were finally rotated upwards and to the left, the eyelids closed, and the patient apparently fell asleep.

In the *second* or *moderate* form, the convulsions commenced as before. After the head had been rotated to the right, and as the clonic spasms were commencing, the fingers of the right hand were drawn in to the palm, the hand was then flexed at the wrist, and the forearm bent to a right angle and placed across the chest. The muscles of the right leg at the same time became rigid, and the foot strongly inverted. Clonic spasms then occurred in the muscles of the arm and fore-arm, the flexors being chiefly affected. A few spasmodic twitchings were to be seen in the leg and thigh, chiefly in the extensors. There was never any flexion of the hip or knee.

In the *third* variety, the convulsions became general. The fit commenced as before, and passed through the various stages enumerated above. After flexion of the right fore-arm, the arm was slowly raised at the shoulder until it was nearly at a right angle with

the body; the tonic spasm then passed to the muscles of the left arm and of the left leg in the following order:—the fingers of the left hand were first drawn in to the palm; the arm was then raised upwards and brought over to the right side, so that the hand approached the forehead; the left leg was at the same time flexed upon the abdomen, the knee being slightly bent, the toes spread out, and the foot flexed at the ankle joint. The tonic spasm soon passed off, and a general clonic spasm of the muscles then occurred, the patient foaming at the mouth and making a cackling noise. As the clonic spasms occurred, the left arm was abducted and placed at a right angle with the body, the under surface of the arm, fore-arm, and hand being uppermost. When the spasm passed to the left arm and left leg, both sides of the face were strongly convulsed.

In the general convulsions, the muscles of the right side of the body were more powerfully contracted than those of the left, and the body tended to turn over towards the right side.

The slight fits occurred every few minutes; the intermediate form frequently; the general convulsions only occasionally. *The sequence of the spasm never varied from that described.*

On May 30th the patient died. On *post-mortem examination* a sharp, lancet-shaped spiculum of bone, about a quarter of an inch in length, was found projecting inwards from the margin of a small oval aperture on the left parietal bone. The exostosis had produced a very limited lesion of the cerebral cortex (see Fig. 19) at a point which very closely corresponds to Ferrier's centre for the platysma.—(See 11, Fig. 16, page 73.)[1]

The limited epileptiform spasms characteristic of Jacksonian epilepsy are of the greatest diagnostic (localising) value, for, with perhaps some exceptions, they show that there is a "discharging" lesion in the motor area of the cerebral cortex; and further, in many cases they indicate the exact position of the lesion.[2]

In order to determine the latter point (the exact position of the lesion), it is necessary to observe the exact mode of commencement of the spasm. Our knowledge of the functions of the different portions of the motor cortex, and of the relationship of these areas or centres

[1] The case is reported in the *British Medical Journal*, vol. ii., 1877, p. 290, and in the *Edinburgh Medical Journal* for August 1878.

[2] See with reference to this point the remarks on p. 85.

to the exterior of the skull, is now so far advanced that if we know the muscle or muscles which are first convulsed, we can, in many cases, indicate with great exactitude, not only the particular centre or mass of grey matter which is discharged, but also the point on the surface of the skull which is superficial to it.

Further, Victor Horsley has shown that localised spasms may be elicited by electrical irritation of portions of the human motor cortex; and that by observing the exact character of these spasms it is possible to identify the portion of cortex which is exposed and irritated.

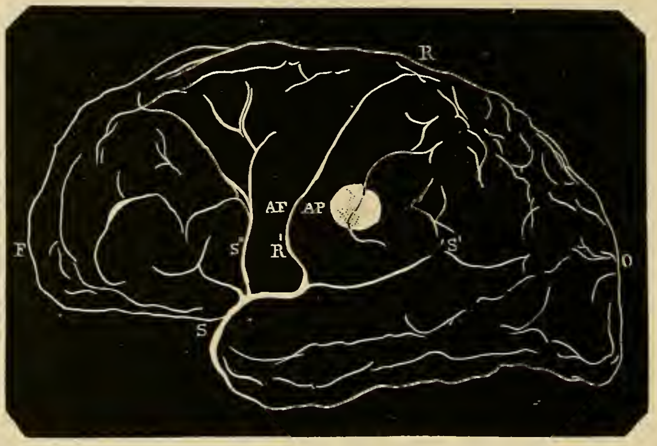

Fig. 19.—Outline of the left hemisphere of the brain in the case of Mary C.

The letter *F* points to the frontal, and *O* to the occipital end of the brain; *S*, *S'* to the fissure of Sylvius; *R*, *R'* to the fissure of Rolando; *A F* and *A P* to the ascending frontal and parietal convolutions. The white circle represents the position of the lesion.

Now, in the great majority of cases, in which localised epileptiform convulsions are due to a lesion in the motor area of the cerebral cortex, the lesion is what we term a "coarse" or naked-eye lesion; and one of the most common forms of localised "coarse" discharging lesion is a tumour. Localised convulsions may, however, be due to a localised and limited softening, or a localised meningitis.

We may therefore say that, when, in addition to the general symptoms of tumour (headache, vomiting, double optic neuritis, &c.), there are localised epileptiform convulsions, the diagnosis of the tumour is strengthened and confirmed; and further, that the position of the tumour is in many cases more or less accurately indicated.

But even in those cases in which the general symptoms of tumour are absent, the presence of localised epileptiform convulsions is suggestive of a tumour; for the absence of general symptoms (headache, vomiting, double optic neuritis) does not necessarily exclude tumour. The point is one of practical importance, for I agree with Dr. Hughlings Jackson in thinking that the frequent recurrence of localised epileptiform convulsions (more especially when they tend to become general, and when the general condition of the patient demands it) calls for operative procedure and excision of the discharging mass of grey matter.

Further, localised epileptiform convulsions are of some value in enabling us to form a *pathological diagnosis;* for undoubtedly the "coarse" lesions, which most frequently involve the motor cortex and act as discharging lesions, are syphilitic and tubercular deposits.

General epileptiform convulsions, in which the spasm simultaneously involves a large number of muscles on both sides of the body, and in which the patient becomes unconscious, may result from a tumour in any part of the intracranial cavity, but are more rare than the localised spasms just described. General epileptiform convulsions of this kind (*i.e.*, in which the spasm is bilateral from its onset) must be clearly distinguished from the generalised epileptiform convulsions which so frequently result from the extension of the discharge in cases of Jacksonian epilepsy. They are probably due either to vascular changes, or to reflex irritation excited by the tumour or by the inflammatory changes in its neighbourhood; in some cases the reflex irritation may perhaps pass to the so-called "convulsive centre" in the medulla; in others, directly to the motor regions of the cortex.

In other cases—but these constitute a small minority—the general convulsions are the result of hæmorrhagic extravasations in or about the tumour.

In some cases the patient dies during the attack—a remarkable difference between the general convulsions associated with tumour and the general convulsions of idiopathic or genuine epilepsy, for in the latter condition a single fit is rarely fatal.

General epileptiform convulsions are of no localising value, unless they have a distinct local commencement, or unless they are preceded by a well-defined and localised aura, indicative of the motor or

sensory area of the cortex which is first discharged, from which the discharge spreads, and in the immediate neighbourhood of which the lesion is in many instances situated. The character of the aura, and the mode of commencement of the spasms, may indicate the position of the lesion, and should therefore in all cases be carefully investigated.

The comparative value of limited paralysis and limited epileptiform convulsions as localising symptoms has given rise to some discussion. The generally accepted opinion is that the former (paralysis) is the more important; for the motor discharge, which produces spasm, may be due to irritation arising at a distance from the portion of grey matter which is discharged; and the position of the centre which is discharged is therefore an uncertain indication of the position of the lesion.

With this opinion, I, for the most part, agree. Certain qualifications are, however, I think, necessary. The discharge which produces well-defined, localised, epileptiform convulsions of distinct Jacksonian type is usually due to the direct irritation produced by a localised "coarse" lesion. It cannot of course be denied that lesions outside the motor area may and do produce localised discharges (either through the vascular alterations which they cause, or reflexly, as described above, see page 78); but I very much doubt if localised epileptiform convulsions, such as I am now referring to, are frequently produced in this way (*i.e.*, by a distant lesion, which does not directly irritate the portion of grey matter, the discharge of which is the cause of the spasm).

Further, a localised paralysis (like a localised convulsion) may be caused by the vascular changes produced by a tumour outside the motor area.

On the other hand, it is important to note that slow destruction of motor grey matter is often unattended with symptoms, *i.e.*, unaccompanied by paralysis; but irritation of motor grey matter (provided that it is sufficiently intense to excite discharge, and that the way out for that discharge is open) of necessity produces spasms.

Again, a localised paralysis may result from a lesion in any part of the neuro-motor apparatus (centres, conductors, peripheral nerves), and it is not always easy to determine the exact point at which that lesion is situated; but localised epileptiform convulsions of the

Jacksonian type are very generally, I believe, due to a discharge of motor grey matter in the cerebral cortex. In rare cases a lesion of the internal capsule is so limited in extent that a localised paralysis, say of the face (as in Hughes Bennett's case), is alone produced; it would be very difficult, and in many cases probably impossible, to distinguish such a paralysis from that due to a cortical lesion.

I am disposed, therefore, to think, on the one hand, that localised epileptiform convulsions of the true Jacksonian type are more certainly produced by a discharging lesion in the motor area of the cortex, than localised paralysis by a destroying lesion. But, on the other hand, I am prepared to grant that when a localised paralysis due to a cortical lesion *is* present, it is more certainly indicative of the exact seat of the lesion than a localised convulsive seizure.

Irritation and discharge indicate that the affected portion of grey matter is situated in the neighbourhood of, rather than at the exact seat of the lesion, while paralysis shows that the affected portion of grey matter is actually involved by the new growth.

Further, it may probably be granted that a lesion outside the motor area is more apt to produce irritation than inhibition of a motor centre—in other words, is more likely to be attended with spasms than paralysis.

Tonic spasms, which most commonly affect the muscles of the back, but which in some cases also involve the muscles of the limbs, and which are in many instances accompanied by retraction of the head, occur in some cases of intracranial tumour. In some cases, the spasmodic contractions are continuous, and produce more or less persistent rigidity; in others, they occur in paroxysms, or the continuous spasms are associated with paroxysmal exacerbations, and a condition not unlike tetanus is produced.

Spasms of this description possess some localising value; for the tumours, with which they are associated, are usually subtentorial, and are often situated in the middle lobe of the cerebellum.

Dr. Hughlings Jackson believes that spasms of this kind are the result of a discharge of grey matter in the cerebellum itself. Dr. Sharkey thinks that the persistent rigidity is due to the effects of irritative pressure upon the pyramidal tracts in the pons Varolii or

medulla oblongata; while he believes that the tetanic convulsive seizures are caused by irritation of grey matter in the floor of the fourth ventricle. I have long taught that a cerebellar tumour may produce tonic spasms, either persistent or intermittent, by pressing upon and irritating the tissues of the pons Varolii or medulla oblongata (both the fibres of the pyramidal tract and the grey matter *throughout* the pons, and not merely the grey matter in the floor of the fourth ventricle). In support of this view, I have been in the habit of mentioning the fact that in large subtentorial tumours, such as tumours of the cerebellum, the pons Varolii and medulla oblongata are usually after death compressed, flattened, softened, and œdematous.

In some cases, tetanic-like seizures of this description are associated with inflammatory changes in the membranes at the posterior part of the base of the brain. In such cases the spasms are, I believe, often due to secondary changes produced in the pons and medulla.

Contractures, Choreic Spasms, &c.

The common forms of contracture, such as are associated with ordinary hemiplegia, may of course be present in those cases in which the tumour involves the motor (pyramidal) tract, and in which secondary descending degeneration results. Hysterical contractures may doubtless occur in those cases of intracranial tumour in which hysterical symptoms are prominent, but no case of the kind has come under my own observation. Possibly, too, persistent spasm and contracture (resembling that produced by Duret in the lower animals) may result from irritation in the dura mater.

Choreic spasms are extremely rare in cases of intracranial tumour. Mills and Lloyd state that in one of Petrina's cases, a glioma involving the substance of both frontal lobes, choreic movements of the right arm were present: in that case tremor in both arms was also a symptom.[1] Wood states that the choreic spasms of cerebral syphilis may assume a distinctly choreic type.[2]

Rhythmical movements, *circus movements*, and *forced movements* are also exceedingly rare. They may be expected to be present in some

[1] Pepper's *System of Medicine*, vol. v., p. 1040.
[2] *Ibid.*, p. 1011.

of the cases, in which the tumour involves the middle peduncle of the cerebellum; the pons Varolii, or Nothnagel's nucleus cursarius in the corpus striatum.

A reeling gait, resembling that due to intoxication, is an important symptom in many cases of cerebellar tumour, as I shall afterwards more fully point out.

The tremor, associated with voluntary movement, which resembles that of cerebro-spinal sclerosis, has already been alluded to.

The condition of the bladder and rectum in cases of intracranial tumour calls for no special remarks. Exactly the same modifications in these reflexes which are met with in other forms of cerebral lesion (such as unconscious urination or retention of urine in coma) may occur here.

Tumours in the lower part of the pons Varolii and in the medulla oblongata seem in some instances to be attended with more decided alterations in the urinary and rectal reflexes than tumours which are situated higher up.

CHAPTER V.

SENSORY DERANGEMENTS—TOUCH—SIGHT—HEARING—TASTE—SMELL.

Disorders of Sensation.

The derangements of sensation which occur in the course of intracranial tumours are (with the exception of headache) usually less prominent and persistent symptoms than the corresponding derangements of motion.

Derangements of the Sensibility of the Skin.

Derangements of the sense of touch are of frequent occurrence, and may be either in the direction of diminished or increased functions.

Anæsthesia.—The extent and distribution of the tactile impairment which may be present is subject to great variations, and depends upon the position of the tumour, and the exact manner in which the anæsthesia is produced.

In some cases, in which the anæsthesia is widely distributed and not very profound, the impairment is due to a general depression of the cerebral (perceptive) centres rather than to any localised derangement of the tactile centres. This form of anæsthesia is frequently met with in the later stages of intracranial tumours. In the earlier stages, anæsthesia due to this cause is seldom considerable, though I have known it sufficiently great to completely mask the pain of a scrofulous affection of the wrist joint.[1]

In other cases the anæsthesia is half-sided in distribution. In such cases the hemi-anæsthesia is usually combined with hemiplegia, and the tumour is (generally) so placed as to involve the posterior third of the posterior division (or sensory portion) of the internal capsule; but it may be situated above or below this point. Large

[1] See a case of scrofulous tumour of the cerebellum, reported in the *Edinburgh Medical Journal* for June 1879, p. 1073.

cortical or subcortical tumours, which involve the sensory fibres of the internal capsule as they pass to the tactile centre of the cortex, may also produce hemi-anæsthesia.

Tumours involving the hippocampal region and the gyrus fornicatus should, if Ferrier and Schäfer are correct in thinking that the centre for touch is situated in these parts of the brain, be attended with hemi-anæsthesia on the opposite side of the body.

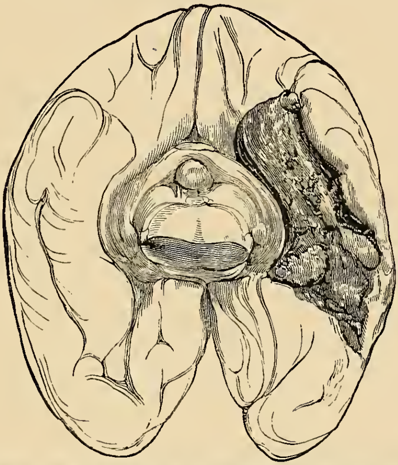

Fig. 20.—The under surface of the left hemisphere of the brain of a monkey, showing a lesion causing complete anæsthesia of the opposite side of the body. (After Ferrier.)

Ferrier places the centre for the sense of touch in the hippocampal region; he found that destruction of this part of the brain in the monkey (see Fig. 20) was followed by hemi-anæsthesia on the opposite side of the body.

Horsley, Schäfer, and Sanger Brown found that destruction of the gyrus fornicatus produced hemi-anæsthesia on the opposite side; and this loss of sensibility seemed to be permanent. But "whether," says Schäfer, "the perception of tactile and general sensibility is confined to the gyrus fornicatus and its continuation into the gyrus hippocampus, can only be effectually cleared up by the entire removal of these parts of the limbic lobe—an operation of the greatest difficulty, but one which I have not given up hopes of ultimately effecting. In the meantime, I would affirm the extreme probability of this hypothesis on evidence obtained by exclusion, for I

have never been able to determine the existence of any permanent diminution of sensibility after operations upon other parts of the cortex, unless there were a possibility of the limbic lobe having been directly or indirectly involved." [1]

Whether tumours which destroy the motor area of the cortex produce any loss of tactile sensibility in the parts which are paralysed is a disputed point. Charlton Bastian and some other authorities think that some degree of anæsthesia is often produced in this way; and I myself have met with cases which seem to lend support to their view.[2]

Alternate hemi-anæsthesia (loss of tactile sensibility in the limbs on the opposite, and in the face on the same side as the lesion) has already been referred to. As previously stated, it is generally accompanied by hemiplegia, and may be caused by a tumour in the pons Varolii.

Localised anæsthesia in the territory supplied by the fifth nerve should be carefully looked for in all cases of intracranial tumour. When present, it is a valuable localising symptom, analogous to the paralysis which is limited to the muscles supplied by a cranial nerve.

Hyperæsthesia (excluding headache, which has already been described) has seldom been a prominent symptom in the cases of intracranial tumour which have come under my notice. The experience of Mills and Lloyd seems different, for they state, "hyperæsthesia occurs so commonly as to be almost regarded as a general symptom of brain tumours. Sometimes it is confined to the head; sometimes it is generally diffused; more frequently it is present in the limb or limbs affected with the paralysis. With hyperæsthesia the patients often complain of spontaneous pain in the limbs." [3]

Neuralgic pains, with or without hyperæsthesia of the face or scalp, are of frequent occurrence. When strictly limited to the area of distribution of the fifth nerve, they are of localising value; and this is more especially the case when all three branches of the nerve

[1] "Experiments on Special Sense Localisation in the Cortex Cerebri of the Monkey." by E. A. Schäfer, F.R.S.: *Brain*, January 1888, p. 379.

[2] See, for example, a case reported in the *Edinburgh Medical Journal*, August 1878, p. 144.

[3] Pepper's *System of Medicine*, vol. v., p. 1042.

appear to be involved; in such cases there is reason to suspect involvement of the trunk of the nerve within the cavity of the cranium.

Derangements of the Sense of Sight.

Derangements of the sense of sight are frequently associated with intracranial tumours. The most common alteration is the dimness of vision (diminished acuity and restricted field), which is so frequently met with in the later stages of double optic neuritis, and with post-neuritic atrophy.

All degrees of impairment, from the slightest loss of visual acuteness up to complete blindness, are met with.

The state of vision in those cases of intracranial tumour in which there is optic neuritis is of so much importance that no apology is necessary for referring to several points which have already been mentioned in a previous chapter.

The fact that in the earlier stages there is often no impairment, either of the acuteness or of the field of vision, has been previously emphasised.

In most cases in which the papillitis remains for any time, more or less impairment, and in some cases total loss of vision, result. This is not always so, for cases are occasionally met with, in which even after well-marked optic neuritis has persisted for a long time (weeks or even months) vision has remained good. In connection with this aspect of the subject, it is important to remember that the inflammation of the optic discs can in many cases be most beneficially influenced by treatment. This is more especially the case when the cerebral lesion is syphilitic. In such cases sight may be perfectly, or almost perfectly, restored after the patient has been completely blind; and some authorities think that even in non-syphilitic cases of brain tumour, double optic neuritis with total blindness may be so completely recovered from, that vision becomes perfect, and little or no perceptible ophthalmoscopic alterations remain. In my own experience, when a patient suffering from cerebral tumour becomes completely blind, in consequence of double optic neuritis, recovery is usually imperfect even in syphilitic cases. In almost all the cases of cerebral tumour, with double optic neuritis and total loss of

vision, which have come under my observation, some defect of vision has persisted (see Figs. 21 and 22), and some alterations in the fundus, which could be detected with the ophthalmoscope, have remained.

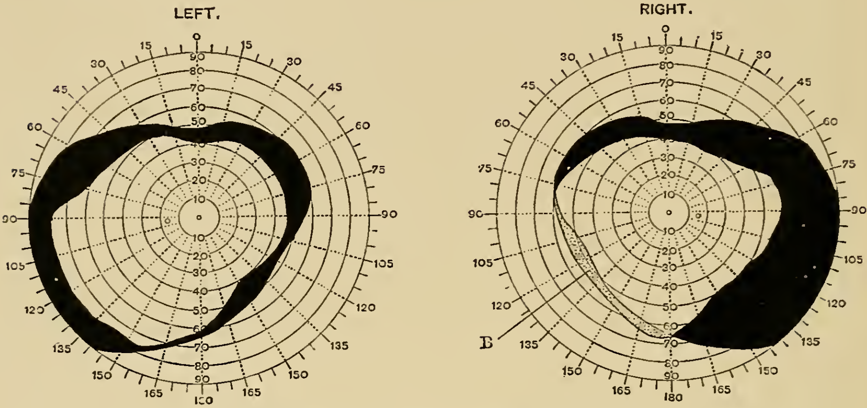

FIG. 21.—Perimeter chart of the fields of vision, showing peripheral constriction, the result of former optic neuritis, in a case of intracranial syphilis.

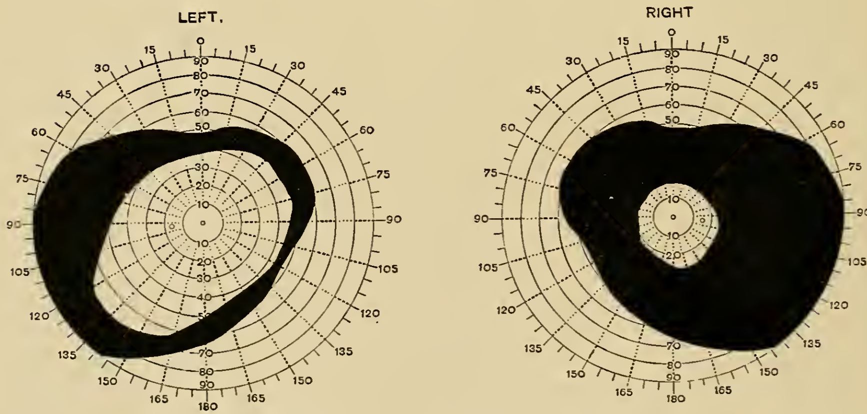

FIG. 22.—Perimeter chart of the fields of vision for white showing marked contraction of the right and some contraction of the left field, the result of old papillitis in a case of intracranial syphilis.

In most cases of optic neuritis in which vision becomes impaired the loss is slowly and gradually established; but in some the loss of sight is rapid or even sudden. In a case of scrofulous tumour of the cerebellum, which I have already referred to, sight and hearing were

both suddenly lost, the patient remaining completely blind and deaf during the whole subsequent period of the case.

In some cases, again, there is temporary loss of vision. With reference to this point Dr. Hughlings Jackson states, "In some cases of optic neuritis the sight fails for a time totally or partially, although at most times it is good. The patient may be able to read the smallest type, and yet occasionally for a few minutes become quite blind."[1]

The dimness of vision which is the result of double optic neuritis in cases of intracranial tumour, since it is due to a peripheral change which may be produced by a tumour in any part of the cranial cavity, has no localising value.

Blindness due to primary optic atrophy is occasionally, but rarely, met with in cases of intracranial tumour. When it does occur, it is usually due to the pressure of the tumour upon the optic chiasma, or upon the optic nerve trunks in front of the chiasma. In one most interesting case, which was sent to me by Dr. Berry, and which is still under observation, bilateral temporal hemianopsia without optic neuritis first developed, and in the course of time was followed by complete blindness and general atrophy of both discs.

Hemianopsia is sometimes met with in cases of intracranial tumour, and is an important localising symptom.

The most common variety—lateral or homonymous hemianopsia —may be due to a lesion (1) in any part of the visual *tract* behind the optic chiasma, optic tract—posterior part of the internal capsule, visual conducting fibres, as they pass through the centrum ovale to join the visual centre in the cortex; or (2) of the visual centre itself, *i.e.*, the grey matter of the occipital lobe.

A lesion (tumour) involving the left visual tract, behind the chiasma, produces blindness in the left side of each retina, or, in other words, is attended with right-sided homonymous hemianopsia. In the case of a left-sided lesion, the patient is consequently unable to see objects on his *right*. *Vice versâ*, a right-sided lesion produces left-sided homonymous hemianopsia.

The exact character of the hemianopsia, as shown by careful perimeter measurements, differs considerably in different cases. In some, the divisional line passes right down through the centre of the

[1] *Transactions of the Ophthalmological Society of the United Kingdom*, vol. i., p. 70.

field, the fixing-point being, however, usually spared.—(See Fig. 23.) In consequence of this fact, central vision in cases of homonymous hemianopsia is usually good.

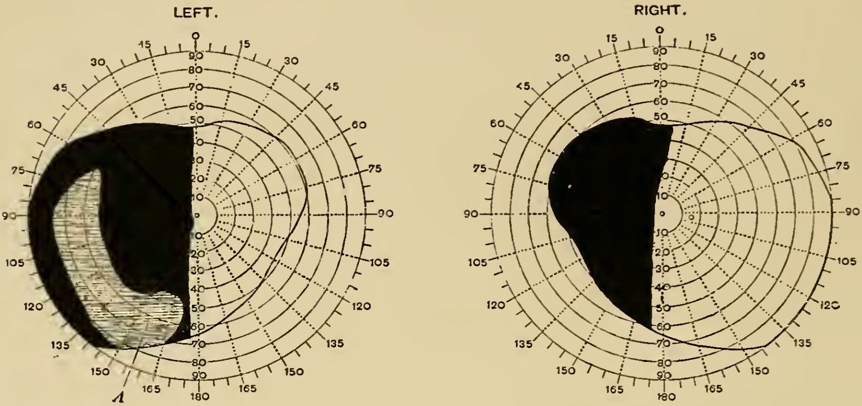

FIG. 23.—Perimeter chart of the fields of vision in a case of left-sided homonymous hemianopsia, which was in all probability due to an old cortical lesion of the posterior part of the right visual centre.

The defect of vision does not, it will be observed, come quite up to the middle line. There is absolutely no contraction of the sound (seeing) half of the field in either eye—a remarkable contrast with the cases represented in Figs. 24 and 25. In the shaded portion of the left half of the left field (to which the letter *A* points) the loss of vision was not absolute.

In other cases the contraction of the visual field is not limited to the side of the hemianopsia, but involves more or less of the peripheral portion of the opposite (seeing) halves of the fields. This peripheral constriction of the seeing half of the retina is usually, so far as I know, greater *on the side of the cerebral lesion.* Thus a lesion in the left hemisphere, which produces right-sided hemianopsia, with peripheral constriction of the left half of each visual field, will produce much greater constriction of the left half of the left field than of the left half of the right.—(See Figs. 24 and 25.)

The exact significance of this peripheral constriction, in the seeing halves of each retina, which is greater in that on the same side as the lesion, is not very clear. It may apparently be caused by a lesion which is situated in the neighbourhood of the internal capsule as well as by a lesion which involves the cortical visual centres. I am disposed, provisionally, to think that when it results from a lesion of the cortical centres, the lesion is situated farther forwards—*i.e.*, more

in the neighbourhood of the angular gyrus—rather than in that part of the visual centre which is situated in the tip of the occipital lobe, and which has been termed the "half-vision" centre.

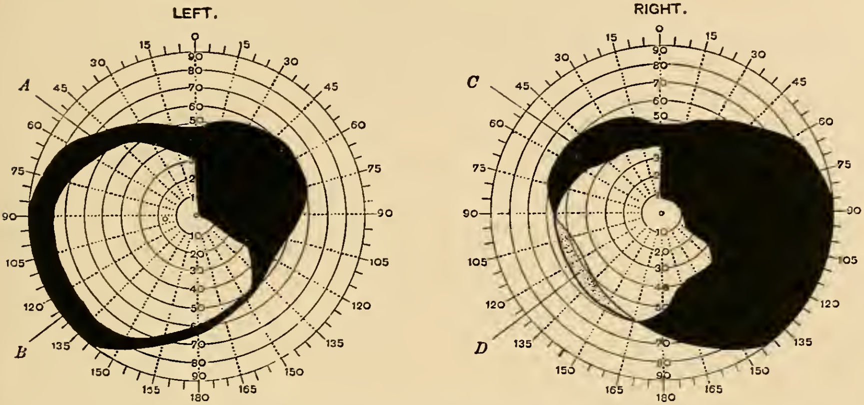

FIG. 24.—Perimeter charts of the fields of vision for white in a case of temporary right-sided hemiplegia and hemi-anæsthesia, showing partial right-sided homonymous hemianopsia; some peripheral contraction of the whole field in the left eye (the parts to which the letters *A* and *B* point); and contraction of the upper part of the left half of the right field (the part to which the letter *C* points). Over the dotted area to which the letter *D* points the field extends beyond the nominal average.

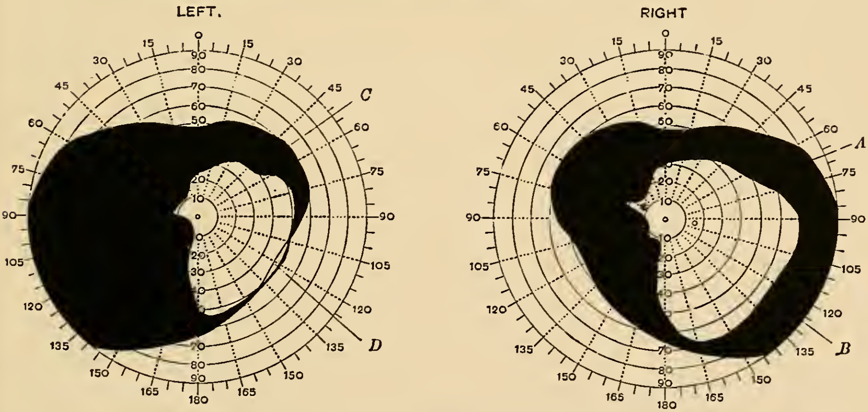

FIG. 25.—Perimeter charts of the fields of vision for white in a case of left-sided hemiplegia, showing left-sided homonymous hemianopsia; considerable peripheral contraction of the whole field in the right eye (the part to which the letters *A* and *B* point); and contraction of the upper part of the right half of the left field (the part to which the letter *C* points).

The exact construction of the visual centre, and the relationship of the two visual centres to the two eyes and to one another, is

undoubtedly very complicated, and not yet thoroughly understood. It seems, however, well established that visual, like all other sensory impressions (with perhaps the single exception of those proceeding to the smell centre), pass up to the opposite side of the brain; in other words, visual impressions of objects on the right hand side of

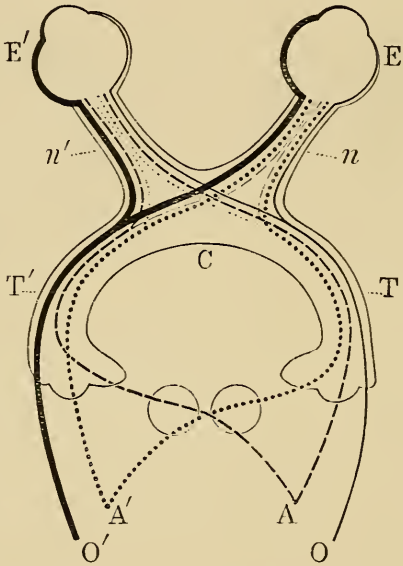

Fig. 26.—Scheme of the optic tracts and visual centres. (After Ferrier.)

A the right, and *A′* the left angular gyrus; *C*, optic chiasma; *E* the right, and *E′* the left eye; *N* the right, and *N′* the left optic nerve; *O* the right, and *O′* the left occipital lobe; *T* the right, and *T′* the left optic tract. The thin continuous line —— represents the retinal relations of *O*; the thick continuous line represents the retinal relations of *O′*; the interrupted line - - - indicates the retinal relations of *A*, and the dotted line ...... the retinal relations of *A′*. The relations of *A* and *A′* with the eye on the same side are indicated by finer interrupted and dotted lines respectively.

the middle line, which are received on the left half of each retina, pass up to the left hemisphere of the brain; visual impressions of objects on the left side of the middle line, which are received on the right half of each retina, pass up to the right hemisphere. In order that this may be effected, the nerve fibres passing from the inner (nasal) half of each retina decussate in the chiasma; in other words, visual impressions which are seen to the right by the right eye, and visual impressions which are seen to the left by the left eye,

decussate; while visual impressions which are seen to the left by the right eye, and those which are seen to the right by the left eye, do not decussate.—(See Fig. 26.)

But further, the fact that in cases of lateral or homonymous hemianopsia there is no loss of central vision, together with the results of experimental observations on the visual centres in the lower animals, seems to show that the visual impressions which impinge on the fixing-point pass up to *each* cerebral hemisphere; and that the visual centres in each hemisphere are connected in a complicated manner.

Temporal hemianopsia (see Figs. 27 and 28) is comparatively rare. In it the inner (nasal) side of each retina is blind, and the patient is unable to see objects on the left of the middle line with the left eye, and on the right of the middle line with the right eye. Since central vision may be unimpaired, and objects on the right of the middle line can be seen by the left eye, and objects on the left of the middle line by the right eye, the visual defect may either be unnoticed by the patient, or it may attract comparatively little attention; in many cases, in fact, it is only discovered when the visual fields are tested by the physician. This form of hemianopsia is almost always due to pressure on the centre of the chiasma; and the cause of the pressure is very generally a new growth of the pituitary body (such as is represented in Figs. 49 and 50), an aneurism of one of the arteries forming the anterior part of the circle of Willis, or a syphilitic gumma in the fore part of the interpeduncular space. Probably in some cases bilateral temporal hemianopsia is due to a localised meningitis involving the centre of the chiasma.

In Fig. 26 the arrangement of the visual fibres suggested by Ferrier is shown. Ferrier believes that the visual centre is very extensive, and is situated both in the angular gyrus and occipital lobe. Schäfer's recent experiments are opposed to some of Ferrier's results. Schäfer did not observe any visual defect whatever, after destruction of the angular gyrus; he found, however, that destruction of one occipital lobe is followed by immediate and persistent hemianopsia, and that the complete removal of both occipital lobes is followed by total and persistent blindness, as Munk also found.[1]

[1] *Brain*, January 1888, p. 363 *et seq.*

Nasal hemianopsia is exceedingly rare. In it the outer half of each retina is blind (see Fig. 29), and the patient is unable to see objects on the left side of the middle line with the right eye, and on the right side of the middle line with the left. Nasal

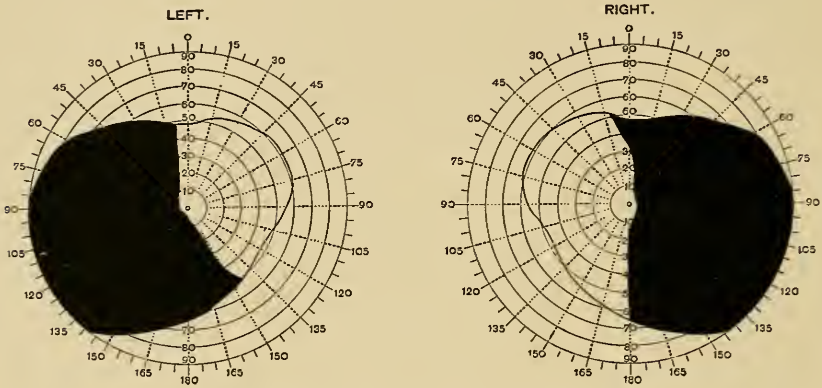

FIG. 27.—Perimeter chart showing temporal hemianopsia in a case of enormous aneurism of the right internal carotid artery.—(See Fig. 55.) The chart was taken by Dr. Berry on November 29, 1883.

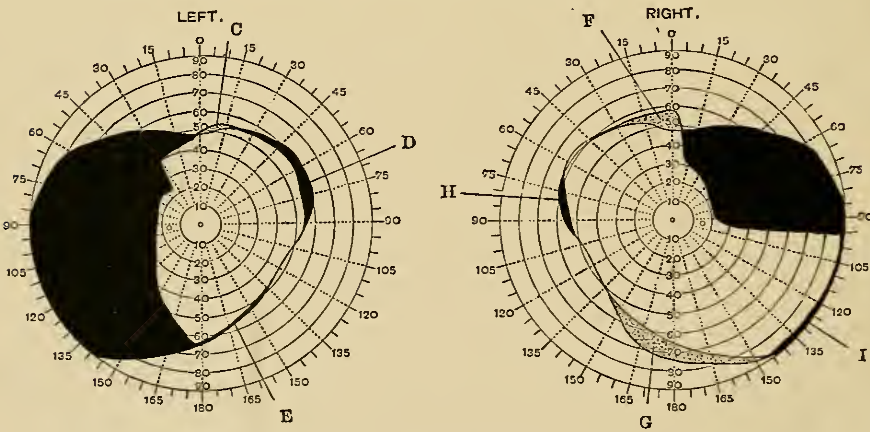

FIG. 28.—Perimeter chart showing partial temporal hemianopsia in a man aged fifty-six. The cause of the condition, which completely passed off under large doses of iodide of potassium, was very obscure. The patient, who denied syphilis, complained of constant dull frontal headache, stupidity, loss of memory, and inability to exert himself. The radial pulse was 84.

hemianopsia can only be caused by a lesion which involves each outer side of the chiasma, leaving the centre and fore part of the chiasma

free. It is theoretically possible that two independent tumours might be so symmetrically placed as to produce such a condition; but, so far as I am aware, no such cases have been met with. It is very unlikely that a single tumour could produce this form of hemianopsia, *i.e.*, could involve each outer side of the chiasma without involving the centre too. This rare condition is usually caused by the pressure of a symmetrical enlargement of each internal carotid artery, or a symmetrically placed focus of inflammation.

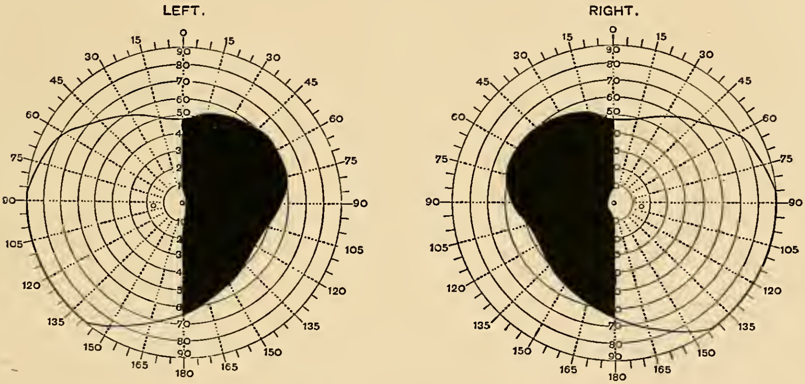

FIG. 29.—Nasal hemianopsia.

Unilateral optic neuritis, with or without dimness of vision, may, as has been previously stated, be due, in rare and exceptional cases, to an intracranial tumour; but in the vast majority of cases in which this condition and in which unilateral optic atrophy occur, the lesion is situated within the cavity of the orbit. When the optic neuritis is due to the last-mentioned cause, some of the muscles of the affected eye are generally paralysed from implication (within the cavity of the orbit) of the nerve trunks which supply them; and in many cases protrusion of the eyeball, or other local evidence of the presence of an intra-orbital lesion, is present.

Temporary lateral or homonymous hemianopsia is common in cases of migraine; and when an intracranial tumour is complicated, as it may be by migraine, this symptom may of course be present.

A discharging lesion of the visual centre in one hemisphere (say a syphilitic gumma on the surface of the occipital lobe) should theoreti-

cally be manifested externally by flashes of light, seen on one side of the middle line, but, as we might expect, usually referred by the patient, to the eye corresponding to that side of the middle line to which the flashes of light are projected. A discharging lesion of the left half-vision centre would, for instance, produce discharge of nerve cells connected with the left half of each retina; and this discharge would be represented in consciousness by a bright light seen on the right side of the middle line, and therefore very usually thought by the patient (ignorant of the construction of the visual mechanism) to be seen with the right eye.

Discharges of this kind are comparatively common in cases of epilepsy, and constitute one of the forms of visual aura; but, so far as I know, they have been very rarely observed as isolated phenomena (*i.e.*, without epilepsy, without being followed by unconsciousness, or unconsciousness and epileptiform convulsions).

Two cases have, however, come under my own observation. In the first—a case of melanotic sarcoma, in which many sarcomatous nodules were scattered throughout the brain tissue—the patient suffered, at short intervals, from paroxysms of violent headache, during which marked vaso-motor flushings, alteration in the pulse frequency, and *sensations of vivid flashes of light in the right eye* occurred. At the autopsy, a large nodule of melanotic sarcoma was found on the surface of the tip of the left occipital lobe (visual centre for objects seen to the right of the middle line), and it is probable that the flashes of light, which were referred to the right eye, were the result of nervous discharges caused by the irritation of this lesion. In that case there was no hemianopsia, so far as could be ascertained by the rough method of examination (*i.e.*, without exact perimeter measurement).

It may be argued that the flashes of light were in this case the result of peripheral (vaso-motor) alterations in the retinæ themselves, for well-marked double optic neuritis was present. But if this had been the case, it is difficult to see why the flashes of light were referred to one and not to both eyes. And again, if the flashes of light were in this case due to local alterations in the retina, why are they not more frequently seen in other cases of cerebral tumour in which optic neuritis is present? A satisfactory answer to this last

question is perhaps to be found in the fact that vaso-motor flushings are not common in cerebral tumours; that in this case these vaso-motor phenomena were very striking, and that the two phenomena (vaso-motor flushings and flashes of light) were always associated together; in other words, the flashes of light never occurred without the flushings. Whether this association was constant in the earlier periods of the case I am unable to say; but the first symptom which the patient experienced was the sensation of a bright light (like the electric light —a globe of bright light) in the right eye.

Now, judging from analogy, a discharging lesion of one half-vision centre ought to produce repeated flashes of light referred to the opposite side of the middle line (or by the patient to the opposite eye); these discharges, which are manifested externally by flashes of light, should not be attended by loss of consciousness, while they should be followed by temporary homonymous hemianopsia—(sensory paralysis in the parts affected with the convulsion)—just as a localised discharging lesion of a motor centre produces localised epileptiform convulsions, which are unattended by loss of consciousness, and which are followed by temporary (epileptiform) paralysis of the muscles which were convulsed.

Further, we might expect that every now and again the sensory, like the motor discharge, would extend to and involve other adjacent and more distant centres; and that the attack would sometimes culminate in loss of consciousness and a general epileptiform convulsion.

Most of these conditions were rigidly fulfilled in my second case, which is reported in the *Edinburgh Medical Journal* for August 1887.

In cases of hemianopsia there is rarely any perceptible difference to be detected by means of the ophthalmoscope between the affected and sound halves of the retina.

It is obvious, from the statement which has just been made, that hemianopsia is an important localising symptom. In all cases, therefore, of suspected intracranial tumour the condition of the fields of vision should be carefully investigated; and in those cases in which any defect in the visual field is detected, an exact outline of the field should be carefully mapped out by means of the self-registering perimeter.

Subjective visual sensations, such as flashes of red light, not infrequently precede an epileptic fit. The facts (1) that the aura is the external manifestation of a discharge of grey matter, and in some cases of a very limited portion of grey matter, in the cerebral cortex; (2) that by observing the character of the aura we can in many cases form an opinion, and in some cases a very accurate opinion, as to the portion of grey matter which is so discharged; and (3) that in cases of "coarse" cerebral lesion the grey matter which is first discharged (*i.e.*, which is represented by the aura) is usually situated in the immediate neighbourhood of the lesion, have been already referred to. They show the importance of accurately noting the exact character of any subjective sensations which may precede epileptiform seizures in all cases of intracranial tumour.

Derangements of the Sense of Hearing.

Deafness is not often caused by an intracranial tumour; when it does occur, it is usually due to involvement of the trunk of the auditory nerve, either by the tumour itself or by the inflammatory products in its neighbourhood. In some cases, loss of hearing is associated with stupor and mental impairment; in other words, it is part and parcel of a general impairment of the cerebral functions, rather than of a lesion of the auditory nerve apparatus in particular. Dr. Hughlings Jackson says, with regard to this point, "It is very striking that whilst tumours of the cerebrum or cerebellum often produce defect or loss of sight of both eyes, although in an indirect way, they never, so far as my experience goes, produce deafness of either side in any way, with the exception of tumours pressing on the auditory nerve, and those producing greatly raised pressure under the tentorium."[1]

The reason why deafness does not result from localised cerebral lesions, such as tumours, may possibly be that the auditory nerve on each side is connected with both cerebral (auditory) centres. We have already seen that, when the visual centre on one side is destroyed, there is hemianopsia on the opposite side, and (according to Ferrier) no loss of central vision. Now, if we were in a position to map out the auditory field in the same accurate

[1] *Transactions of the Ophthalmological Society*, vol. i., p. 78.

manner that we can map out the visual field, it is quite possible that in cases of destruction of one auditory centre we might find that while "central hearing," so to speak, is retained, there is some peripheral limitation of the auditory field. With the object of discovering such defects, the systematic examination of the patient's capability of hearing sound vibrations of different range might perhaps be more thoroughly employed than is usual at present. Further, we know that destruction of a portion of the left auditory centre may produce (in right-handed persons) a special form of loss of hearing, *i.e.*, "word-deafness;" just as destruction of a portion of the left visual centre may produce "word-blindness." And Ferrier has shown that, while destruction of one auditory centre (see Fig. 30) does not produce one-

FIG. 30.—Outline of the brain of a monkey, showing the position of the lesion causing loss of hearing. (After Ferrier.)

The dotted line indicates the extent of the surface exposed by removal of the skull.

The corresponding part of the opposite hemisphere was also destroyed.

sided deafness, yet complete destruction of both auditory centres does produce total deafness in both ears.

Just as this chapter was going to press, Schäfer's paper on Special Sense Localisation appeared. The results which he records are very different from those obtained by Ferrier. Schäfer did not obtain in any of his experiments unmistakeable evidence, nor indeed any evidence at all, of the impairment or abolition of the senses of hearing, smell, or taste.[1] And he states, "I believe, therefore, I am justified in asserting that the supposed localisation of the auditory

[1] *Brain*, January 1888, p. 262.

perceptive faculties in the temporal lobe in monkeys has no experimental evidence in its favour, and the case I have described, in which both temporal lobes (including the corona ammonis) were wholly removed without any permanent diminution in the acuteness of hearing, bears strongly against that view."

It is not perhaps unreasonable to suppose that the plan of the auditory and visual nerve mechanisms may be somewhat similar, and that if we could examine the condition of hearing in the same accurate and complete manner, and with the same facility that we can examine the condition of vision, we might find that some defect in hearing, analogous to hemianopsia, does directly result from cerebral tumours. But in the present uncertainty as to the position of the auditory centre, it is impossible to make any definite statement on the matter.

The fact that deafness is so rarely caused by intracranial tumours, while loss of vision (due to optic neuritis and post-neuritic atrophy) is so common, seems to me an argument against the view that the double optic neuritis of intracranial tumours is due to a neuritis descending *along the nerve fibres*, though it does not tell in the same strong way against an inflammatory process descending, *along the membranes*, to the vaginal sheath, and thence to the intravaginal portion of the optic nerve.

Ross states that "the injections of E. Weber have shown that there is a communication between the arachnoid cavity and the labyrinth by means of the aqueduct of the cochlea; and consequently increased intracranial pressure may produce an affection of the auditory apparatus, similar to that which occurs in the eye under the same circumstances." I do not know on what clinical grounds he bases the latter part of this statement, but the case of cerebellar tumour (to which I have previously referred), in which both hearing and sight were suddenly lost, in consequence apparently of a rapid increase in the intracranial pressure, seems to lend some support to this view, Be that as it may, loss of hearing is very rarely indeed associated with increased intracranial pressure; the case just referred to stands almost alone; and a neuritis of the end-organ of hearing, with resulting deafness, is practically unknown in cases of intracranial tumour. This fact has, however, little force as an argument against the increased pressure theory of the causation of optic neuritis, for any

communication which does exist between the arachnoid cavity and the labyrinth, through the aqueduct of the cochlea, must be very much less free than the communication between the arachnoid and the vaginal space surrounding the optic nerve; and it has yet to be shown that the terminal expansion of the auditory nerve in the labyrinth is constructed so as to be injuriously affected (*i.e.*, inflamed), even if increased intracranial pressure were brought to bear upon it in this way. The theory is nevertheless plausible, and, if we may suppose that the communication between the arachnoid cavity and labyrinth were exceptionally free, it is perhaps possible to account in this way for the very remarkable and quite exceptional total deafness in the case of cerebellar tumour to which I have just referred.

In more than one of my cases of cerebral tumour the tuning-fork did not appear to be heard, when placed over certain limited areas of the skull, which, on post-mortem examination, were found to be abnormal—in one case unusually thin, and in another unusually thick, owing to periostitis. Whether this apparent non-conduction of the skull-sounds only existed in the imagination of the patient, or whether it was real, and if it did actually exist, whether it has any definite and localising value, I am unable to say. But the point is perhaps worthy of future investigation.

Disorders of the Sense of Smell.

Derangements of the sense of smell are rarely met with in cases of intracranial tumour; when present, they are usually due to direct involvement of the olfactory nerves or olfactory bulbs by the tumour or the inflammatory changes in its neighbourhood.

The position of the centre for smell is not yet definitely determined. Ferrier places it in the hippocampal lobule (see Figs. 31 and 32); but Schäfer was unable to detect any impairment of the sense of smell even after cutting away the antero-inferior extremity of the temporal lobe on both sides.[1]

It is probable that if we had the means of accurately testing the range and field, so to speak, of the olfactory sense, impairment of the sense of smell would be discovered in some cases of cerebral tumour.

[1] *Brain*, January 1888, p. 378.

But whether destruction of any localised portion of the brain in man produces loss of smell in the nostril on the same side, or indeed on either side, has not as yet been demonstrated; but the fact that in some cases of hysterical hemianæsthesia smell seems to be abolished

Fig. 31.

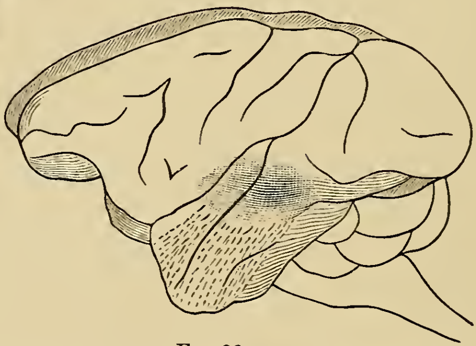

Fig. 32.

Figs. 31 and 32.—Lesions of the right and left hemisphere, causing loss of taste and smell. (After Ferrier.)

In the right hemisphere (31) the shading indicates the extent of destruction of the grey matter. In the left (32) the dark shading indicates the superficial extent of the wound; and the dotted lines the extent of internal destruction of the lower portion of the temporo-sphenoidal lobe.

in the opposite nostril, would appear to show that in man olfactory impressions do cross, as all other sense impressions seem to do, in making their way from the periphery to their cerebral (cortical) centre. The effect which lesions (tumours) involving the anterior commissure have on the sense of smell has not perhaps as yet been sufficiently accurately determined to permit of any definite statement.

Disorders of the Sense of Taste.

Derangements of the sense of taste have rarely been noted in cases of intracranial tumour, and we want more information before

we can speak definitely either as to their occurrence, or as to the position of the lesions which are likely to produce them.

The usually accepted view is that the glosso-pharyngeal nerve is the nerve of taste for the back part of the tongue, and the chorda tympani for the front part.

According to this view, subtentorial tumours (which either directly implicate, or which produce inflammatory lesions involving the trunk of the glosso-pharyngeal nerve at the base of the brain, or its nerve nucleus in the medulla oblongata) should produce loss of taste in the back of the tongue.

Dr. Gowers, however, disputes this view. He believes "that taste-impressions reach the brain solely by the roots of the fifth nerve; and that the doctrine that the roots of the glosso-pharyngeal nerve have anything to do with taste is a curious physiological myth, due to too wide an induction from certain anatomical facts, and from dubious experiments on animals. It is possible," he adds, "that the nerve fibres for taste on the back of the tongue may be *distributed* with the glosso-pharyngeal, reaching them from the otic ganglion of the fifth by the small petrosal nerve and tympanic plexus. This explains the remarkable fact pointed out by Urbantschitsch, and which I have several times observed, that taste may be lost on the back as well as on the front of the tongue in consequence of caries of the walls of the tympanum." [1]

The exact position of the cortical centre for taste is undetermined. Ferrier did not succeed in differentiating any special region related to the sense of taste, but thinks, from the facts which he did observe, that it is in close relation with the olfactory centre (*i.e.*, the lower portion of the temporo-sphenoidal lobe, or hippocampal lobule.—(See Figs. 31 and 32). Schäfer entirely disputes this view.

It is probable, I think, that a more careful and systematic examination of the senses of smell and taste than is at present usually adopted will show that this, like the other senses, is modified in some cases of intracranial tumour.

[1] *Diseases of the Brain*, pp. 25-26.

CHAPTER VI.

MENTAL ALTERATIONS—APHASIA—APOPLECTIC ATTACKS—VISCERAL DERANGEMENTS.

Mental Alterations.

It is probable that, in the great majority of cases of intracranial tumour, some impairment or derangement of the mental faculties is present.

In many cases the mental alteration is so slight that it can only be recognised by those who were intimately acquainted with the patient before his illness, and who are therefore able to detect slight differences, and to accurately compare the present with the previous (normal) mental state. It is unnecessary to say that the friends and relatives of the patient, who are in a position to make this comparison, often attach little or no importance to slight mental differences, even if they notice them.

In other cases the mental changes are so striking as to attract the attention of even the most casual observer.

The mental symptoms and alterations which may be present in cases of intracranial tumour are so varied and numerous, that it is difficult to arrange and classify them, or to give an adequate description of them in a few lines. Looking at the matter broadly, it is possible to group the mental symptoms under one or other of the two great primary divisions into which we have grouped all nerve symptoms, viz., irritative or paralytic; in other words, into symptoms which are associated with exaltation, or, perhaps better, with *ap, arent* exaltation on the one hand, and with diminution of function on the other. The fact that in some cases, for instance, in which the symptoms are suggestive of increased function, as in some cases of mania, the primary lesion is in reality paralytic, has already been referred to (see page 8).

Amongst the former (*positive* symptoms, or those which are usually associated with exaltation of function, but in which the

primary cause is in many instances a "destroying" and not a "discharging" lesion), "alterations in mental disposition," irritability

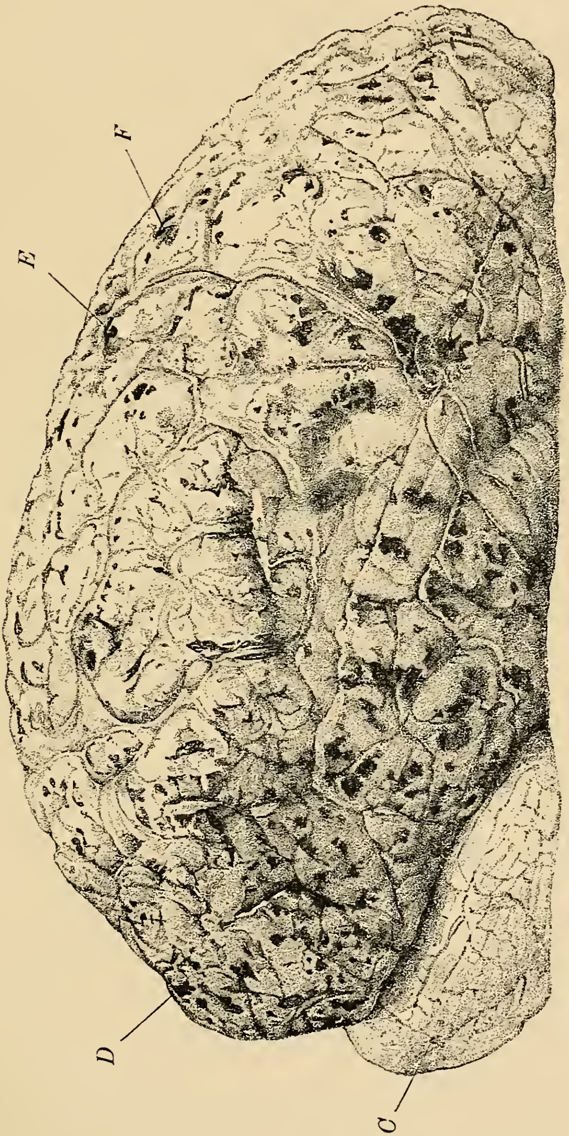

FIG. 33.—Lateral view of the right side of the brain and cerebellum in case of diffused melanotic sarcoma. (Reduced from a photograph.)

The letter *C* points to the cerebellum, on the surface of which no melanotic deposits are to be seen; *D*, *E*, and *F*, to melanotic deposits on the surface of the cerebrum. The whole surface of the brain is studded with similar deposits.

and unevenness of temper, crossness, the liability to be upset by trifles, oddness of manner or address, obstinate wakefulness, many

emotional and so-called hysterical symptoms, and (in rare cases) delusions, hallucinations, threats or acts of violence, outbursts of excitement or attacks of acute mania, may be mentioned.

Amongst the latter (*negative* symptoms, or those which are associated with diminution or abolition of function, and in which the primary lesion is usually, at all events, a "destroying" one), the following are some of the more important:—more or less intellectual impairment, loss of memory, want of attention, loss of the power of

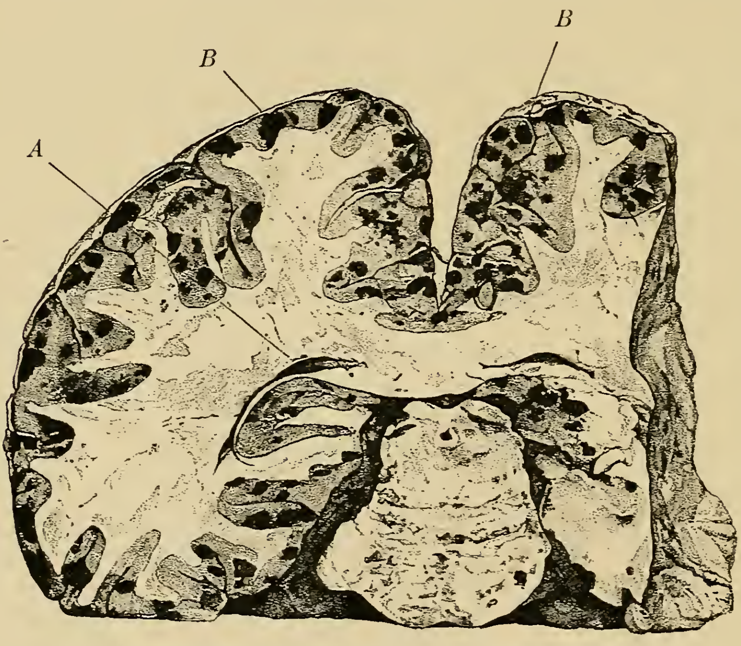

FIG. 34.—Transverse vertical section through the left and portions of the right hemisphere of the brain, at the level of the greatest convexity of the pons Varolii, in case of diffused melanotic sarcoma, showing an enormous number of melanotic deposits in the grey matter of the cerebrum and in the substance of the pons Varolii. (Reduced from a photograph.)

The letter *A* points to a spot just above the lateral ventricle on the left side; *B* and *B*, to melanotic deposits in the grey matter of the cerebral cortex.

mental application and concentration, want of former mental vigour and liveliness, listlessness, taciturnity, depression of spirits, marked melancholia, apathy, lethargy, absence of intellectual interest in surrounding objects, want of attention to calls of nature, excessive sleepiness, stupor,—in short, complete dementia and coma.

The causation of the mental symptoms.—In many cases the mental

alterations are probably due to a widespread derangement of brain function—in others, to localised lesions in special parts.

The alterations in the vascular and lymph supply, and the compression of the nerve tissue, which attend increased intracranial pressure; the fine irritative changes, which in some cases are so extensively diffused through the brain, and which are perhaps the result of some irritative material produced by the metabolism in the tissues of the tumour, or in the area of cerebral softening which

Fig. 35.—Camera lucida drawing of a microscopical section through a portion of three convolutions of the brain in case of diffused melanotic sarcoma, showing numerous sarcomatous nodules in the grey matter. Very low power. (Hartnack, ocular 2, objective 1, and drawing reduced from 8 to 4 inches.)

The letters *a*, *a*, *a* point to the cut edges of the section; *b*, *b* to transversely divided vessels in the membranes on the surface of the brain; *c*, *c* to masses of melanotic sarcoma in the grey matter; *d*, to the white substance in the centre of a convolution.

surrounds it, and the œdema of the brain tissue which attends dropsy of the ventricles, are obvious causes of deranged nutrition or of functional disturbances in the nerve cells of the cortex and in the extremely delicate network of nerve fibres which connects different nerve cells and groups of nerve cells together. It is obvious that even slight derangements in the nutrition of these structures must

necessarily, when widely spread, be attended with some mental alteration, although the symptoms may in some cases be so slight as to be with difficulty recognisable.

FIG. 36.—Camera lucida drawing of a microscopical section of a portion of the grey matter of the cortex of the brain in case of diffused melanotic sarcoma, showing one of the melanotic deposits under a lower power. (Hartnack, ocular 3, objective 4, tube drawn out, and drawing reduced from 7 to $5\frac{1}{2}$ inches.) The pigmented sarcomatous cells (the individual outlines of which it is difficult to distinguish under this magnifying power) are grouped in a most remarkable manner around the blood-vessels.

The letter *A* points to the membranes on the free surface of the convolution; *B*, to a portion of the grey matter which is not invaded; *C*, *C*, to masses of pigmented cells surrounding the vessels.

Again, the localised irritation or destruction which is the direct result of the pressure of the tumour on special parts of the brain tissue, or of the inflammatory and other changes which are estab-

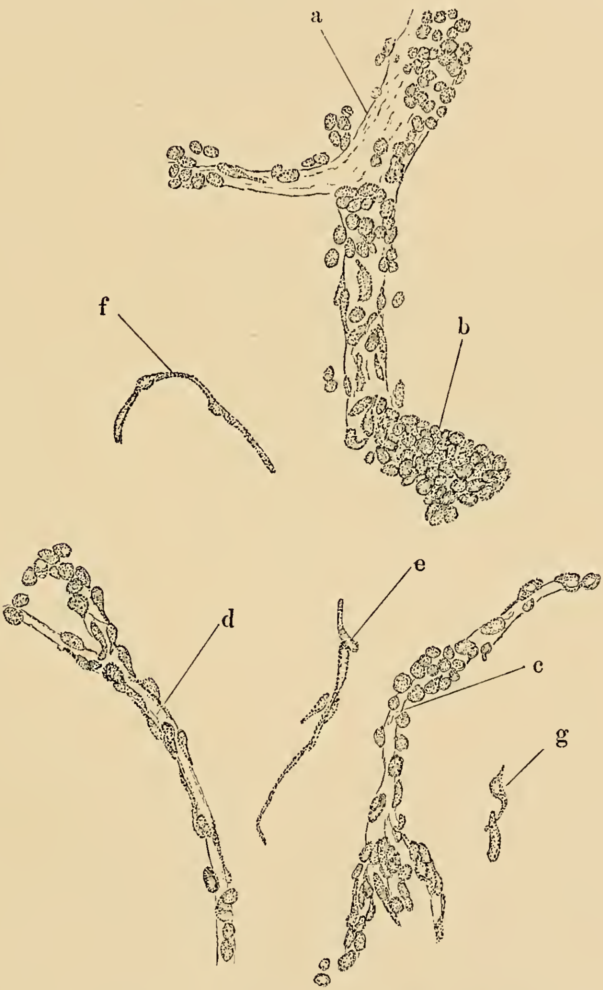

Fig. 37.—Camera lucida drawing of blood-vessels from the grey matter of the brain in case of diffused melanotic sarcoma, showing vessels and pigmented sarcomatous cells surrounding them. (High power—Hartnack, oc. 3, obj. 8, tube out, and drawing reduced from $6\frac{1}{2}$ to 5 inches.)

The letter *a* points to a vessel of some size at a point where the vessel wall is unsurrounded by cells; *b*, to a large mass of cells surrounding a vessel; *c* and *d*, to small vessels surrounded with cells; *e*, *f*, and *g*, to pigmented sarcomatous cells of various shapes, which are making their way along narrow lymphatic spaces, and are in consequence very finely drawn out.

lished in the immediate neighbourhood of the new growth, may cause mental symptoms, either by the direct damage which they occasion, or by the indirect (reflex) effects (irritation and inhibition) which they produce in other and distant parts of the brain tissue. In the remarkable case of melanotic sarcoma which is represented in Figs. 33, 34, 35, 36, 37, and 38, the profound mental disturbance which was present, was obviously due to the extraordinary manner in which

Fig. 38.—Camera lucida drawing of a section through the surface of a convolution of the brain in case of diffused melanotic sarcoma. (High power—Hartnack, oc. 3, obj. 8, tube out, and drawing reduced from $2\frac{1}{2}$ to 2 inches.)

The letter *a* points to the membranes on the free surface of the convolution; *b*, to a small (capillary) vessel passing into the grey matter; *c*, to a group of large, rounded, and in some cases multinucleated sarcomatous cells containing pigment.

the new growth was diffused throughout the whole grey matter of the cerebrum.

Direct damage to the frontal lobe seems in some cases to be attended with distinct mental disturbances, the exact nature of which will be afterwards described. Tumours of the non-motor regions at the back of the brain are, according to Dr. Hughlings Jackson, more frequently attended with mental symptoms than

tumours in the motor area; but whether the mental symptoms, which seem to be associated with lesions of the occipital and other sensory centres, are due to *direct* destruction of tissue (*i.e.*, of the nerve centres of the occipital lobe), or, as is perhaps more likely, to the more widely distributed *indirect* effects (irritative or inhibitory) which a lesion of a sensory centre may be expected to produce upon other centres, is perhaps doubtful. Be that, however, as it may, the mental symptoms which have been described in the foregoing section can hardly be said, in the present position of our knowledge, to have any distinct localising value.

Derangements of Speech—Aphasia.

Any of the different speech derangements, which are classed together under the general term aphasia, *may* be caused by an intracranial tumour, but in most cases the speech derangement is either temporary (post-epileptic aphasia) or incomplete; it is rare to find a tumour producing the more complete forms of permanent aphasia, such as result from embolic plugging of the nutrient vessels.

Aphasic symptoms, when they do occur, are not only of great scientific interest, but of distinct localising value. Hence, in all cases in which speech derangements of an aphasic kind are present, the exact form of the aphasia (whether sensory or motor, word-blindness, word-deafness, aphemia, or agraphia) and the degree of the impairment must be carefully investigated.

As we all know, the speech derangements, grouped together under the common term aphasia, are, in right-handed persons, almost always due to lesions in the left hemisphere of the brain; while in left-handed persons the reverse is usually the case. In those exceptional cases in which in a right-handed person the lesion is found in the right, or in which in a left-handed person it is found in the left hemisphere, the patient may have been ambidextrous; for the few instances in which this is not so, no explanation (unless a second lesion has been overlooked) is at present forthcoming. The presence, therefore, of any distinct degree of aphasia is, in the case of a right-handed person, strongly suggestive that the tumour is situated in the left hemisphere of the brain. Further, if the aphasia is motor

(aphemia or agraphia), the lesion is probably situated in or about the posterior end of the lower or third frontal convolution. In cases of "word-deafness" it probably involves the upper or first temporo-sphenoidal convolution; and in the cases of "word-blindness," it is probably situated in or about the angular gyrus (see Fig. 39). But as previously pointed out, marked and permanent aphasia, whether motor or sensory, is rarely met with in cases of cerebral tumour.

It must also be remembered that aphasia may be due to interrupted conduction through the fibres, which connect the sensory

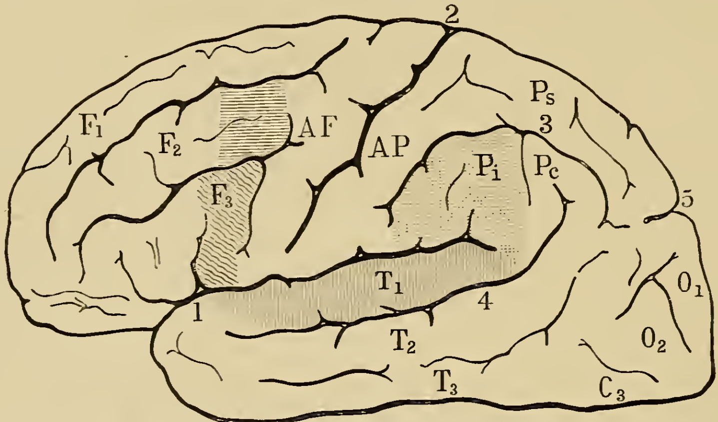

FIG. 39.—Outline of the left hemisphere of the brain, showing the position of the lesion in different forms of aphasia. (After Bernard.)

1. Fissure of Sylvius; 2. Fissure of Rolando; 3. Interparietal sulcus; 4. Parallel fissure; 5. External perpendicular fissure.

*F*¹, first; *F*², second; and *F*³, third frontal convolutions.

AF, ascending frontal; and *AP*, ascending parietal convolutions. *P*, superior-parietal lobule; P¹, inferior parietal lobule, or angular gyrus; *P*², Pli courbe; *T*¹, first; *T*², second; and *T*³, third temporal convolutions. *O*¹, first; *O*², second; and *O*³, third occipital convolutions.

MF, the position of the lesion in motor aphasia (aphemia).

Ag, the supposed position of the lesion in agraphia.

WD, position of the lesion in word-deafness.

WB, position of the lesion in word-blindness.

with the motor speech centres, and it is probable that this is the position of the lesion in some cases of combined sensory motor aphasia. Further, as Dr. Charlton Bastian has shown, disorders of the sensory speech centres necessarily produce more or less derangement of the motor speech centres, which act in conjunction with them. It is impossible to go into details, and to attempt to explain this and the many intricate questions connected with this most interesting and

difficult subject. Enough has been said to show that the exact character of the aphasic symptoms may give important information as to the position of the tumour.

Apoplectic and pseudo-apoplectic attacks.

Sudden losses of consciousness are not uncommon in the course of intracranial tumours, more especially when the new growth is a glioma or a syphiloma.

In some cases the attack is due to hæmorrhagic extravasation in or about the tumour. In these cases, in which the tumour is very often a highly vascular glioma (see Figs. 40, 41, and 42), the behaviour of

Fig. 40.—Extreme dilatation of the blood-vessels in a case of glioma. Several aneurismal dilatations are seen.
The letter *A* points to a portion of a vessel seen in transverse section; its coats are very much thickened.

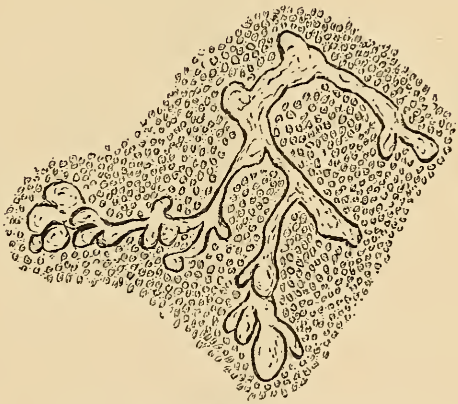

Fig. 41.—Section through a portion of a gliomatous tumour, showing a varicose and aneurismally dilated vessel surrounded with gliol cells.

the temperature during the stage of coma, and the whole progress of the case, may exactly resemble an attack of ordinary cerebral hæmorrhage.

If the patient should die during the stage of coma, the true significance of the post-mortem appearances may under such circumstances be easily overlooked, and the fact that the primary and real lesion is a new growth (glioma or other vascular tumour) may not be recognised. Repeated small hæmorrhages, each attended with coma, of longer or shorter duration, sometimes occur. In the *Edin-*

burgh Medical Journal for January 1887, I have reported a typical case of this kind, in which the remains of several small, old hæmorrhages were found post mortem.

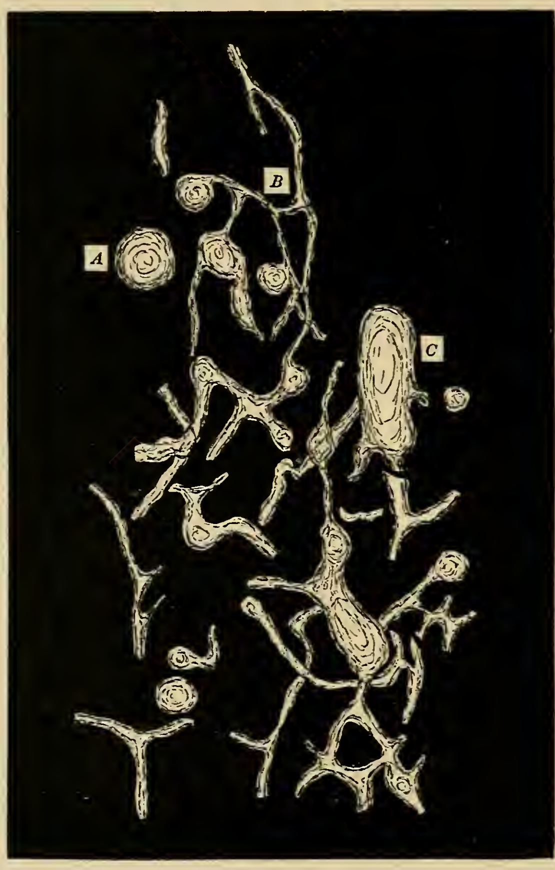

FIG. 42.—Camera lucida drawing of a microscopical section in a case of glioma, showing aneurismal dilatation of the minute vessels. (Stained with picro-carmine, cleared with absolute alcohol and oil of cloves, and mounted in xylol balsam.) Magnified—Hartnack, oc. 3, obj. 8, and drawing reduced from 8 to 4 inches.

The letter *A* points to a transversely divided vessel ; *B*, to a vessel with numerous aneurismal dilatations ; *C*, to a large aneurismal dilatation. The tissue of the tumour, in the midst of which the vessels lie, and many of the vessels themselves, have been omitted from the drawing.

In other cases, in which there is no hæmorrhagic extravasation, congestion, œdema, obstruction of vessels, rapid increase of ventricular dropsy, and an epileptic fit, are possible causes of the coma. Pseudo-

apoplectic attacks, as they are technically termed, are especially common in cases of cerebral syphilis.

State of Nutrition—Temperature—Pulse.

The general state of nutrition is often remarkably well preserved in cases of intracranial tumour; even in cases of scrofulous tumour the general condition may, for a time at all events, be little impaired.

In those cases in which pain, sleeplessness, and vomiting are prominent symptoms, the general health may fail, and emaciation rapidly take place even in the earlier stages of the case. In most cases in which nutrition fails early the condition is due to the presence of associated lesions in the abdominal or thoracic viscera. Towards the end of long-continued cases emaciation is often extreme. In cases of tumour of the pituitary body, the patient often becomes abnormally fat; whether this is due to the lesion of the pituitary body itself, or to the secondary alterations which a tumour in this situation is apt to produce in the surrounding nervous structures, is not as yet determined.

In some cases of intracranial tumour *the appetite is voracious;* and it is perhaps owing to this fact that the body weight is sometimes so well preserved. Drs. Lawson and Bevan Lewis, who, so far as I know, were the first to direct prominent attention to this subject, thought that the voracious appetite was an early symptom in cases of intracranial tumour. Though I have personally met with a voracious appetite in the early stages of intracranial tumour, I am disposed to think that it is much more frequently a late symptom; and that it is usually a result of the general mental deterioration which is so often present.

The temperature is almost always normal or subnormal in uncomplicated cases of intracranial tumour. Elevation of temperature usually indicates the occurrence of inflammatory complications in the membranes (meningitis), but it may be due to cerebritis. Mills and Lloyd conclude, as the result of their observations, that in brain tumours the average temperature of the whole head is elevated several degrees above the normal, and that the elevation of temperature is usually greatest at the station nearest the new growth.[1] I

[1] Pepper's *System of Medicine*, vol. v., p. 1037.

have no observations of my own to offer on this point, but it is obviously one which demands careful investigation. Whether, too, tumours in special parts of the brain produce modifications in the body heat (elevations or depressions)—whether, in short, there are cortial heat centres or not, and, if so, what is the exact position—is still undetermined. On theoretical grounds we might perhaps expect lesions (tumours) in the pons Varolii or medulla oblongata to be attended with more marked temperature alterations than tumours in other parts of the brain tissue; but the theoretical questions, which I have discussed elsewhere,[1] need not be debated here. Lesions in the pons Varolii do, however, appear in some instances to be attended with hyperpyrexia.

In exceptional cases of intracranial tumour, the onset of the attack is attended with febrile disturbances,—probably due in most cases to meningitis, and in some perhaps to tubercular meningitis. In the later stages, rises of temperature due to this cause are more common.

When hæmorrhagic extravasations occur from the vessels of a tumour, the same alterations in the temperature curve may be noted as in cases of ordinary cerebral hæmorrhage (ordinary hæmorrhagic apoplexy).

The exact observation of the temperature is probably of considerable importance in distinguishing the pseudo-apoplectic seizures, which occur in the course of some cases of cerebral tumour, more especially in cases of cerebral syphilis, from the apoplectic attacks associated with hæmorrhagic extravasation. In pseudo-apoplectic attacks, the initial depression which attends the onset of a copious cerebral hæmorrhage is not observed.

In those cases of intracranial tumour in which the temperature is elevated, the observer must of course be on his guard to exclude all other sources of pyrexia (such as inflammatory complications in the thorax or abdomen) before coming to the conclusion that the elevation of temperature is due to the intracranial lesion (meningitis, cerebritis, &c.).

The pulse frequency is, as a rule, either normal or diminished in the earlier stages of intracranial tumours; towards the end it may become increased.

[1] *Practical Medicine and Medical Diagnosis*, p. 68.

Diminished pulse frequency is often associated with increased intracranial pressure. In some cases it is perhaps the direct result of irritation of the vagi nerves, or their nuclei in the medulla oblongata.

Exacerbations of the headache may be accompanied by diminution in the frequency of the pulse.

Vaso-motor flushings are occasionally present; and in one case of melanotic sarcoma, which has been already referred to, they were very striking phenomena. In that case the vaso-motor disturbances were accompanied with severe headache, flashes of bright light in the eyes, blindness, and alterations in the frequency of the pulse.[1]

Alterations in the urinary secretion (other than those depending upon the disturbances of the urinary reflex, which have been previously referred to) are occasionally observed.

The presence of an *excess of phosphates* is very common, but of no localising significance.

Polyuria, glycosuria, and occasionally *albuminuria,* have been noted, more especially in those cases in which the tumour involved the pons Varolii or medulla oblongata. Tumours of the pituitary body seem, in some instances at all events, to be attended with polyuria and glycosuria.

Bed sores are often, of course, present in the later stages of intracranial tumours. *Derangements of respiration* may be the direct result of the intracranial lesion, more especially when it is subtentorial and involves the medulla oblongata, but are usually due to some associated lesion within the thorax. *Cheyne-Stokes* respiration is not uncommon before death.

[1] See *Edinburgh Medical Journal,* July 1887.

CHAPTER VII.

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS.

Diagnosis.

The diagnosis of intracranial tumours is in some cases easy; in others most difficult; in others, again, impossible.

As Dr. Hughlings Jackson—to whom we are indebted for so much of our knowledge on the whole subject of intracranial tumours—long ago pointed out, three questions present themselves for solution, viz.:

1. Is there an intracranial tumour?
2. If so, where is it situated?
3. What is its pathological nature?

Step No. 1.—*Is there an intracranial tumour?*

In the *first* of the clinical groups into which I have divided cases of intracranial tumour a diagnosis is obviously impossible.

In the *second*, it is generally possible to come to a positive opinion without much difficulty; but it is necessary to exclude certain conditions, such as Bright's disease and lead poisoning, in which headache, vomiting, double optic neuritis, and the other "general" symptoms of intracranial tumour may be present.

In the *third*, the diagnosis is still more easily and certainly arrived at; for the presence of symptoms indicative of a local cerebral lesion confirms very strongly the diagnosis of intracranial tumour suggested by the general symptoms.

The cases comprised in the *fourth* clinical group present the greatest difficulties in diagnosis.

(*a*.) The uncertainty and difficulty are in some cases due to the fact that some symptom or symptoms which one would expect to be present in a typical case of intracranial tumour are altogether absent or only slightly marked. Now double optic neuritis is, as we have seen, the most important of all the symptoms of intracranial tumour; and it is in the absence of double optic neuritis that doubt is chiefly

apt to arise. Such conditions as migraine, hysteria, hypermetropia, and anæmia with or without hypermetropia, in which many of the general symptoms of an intracranial tumour may be present, are those which most frequently give rise to difficulty.

(*b.*) In other cases the symptoms are clearly indicative of a local cerebral lesion, but the general symptoms are not characteristic of a tumour.

Repeated attacks of Jacksonian epilepsy—(1) without headache, vomiting, and double optic neuritis; or (2) with slight headache, and without vomiting and optic neuritis—may be mentioned in illustration. The lesion in such cases *may* be a tumour, but without cutting down upon it and actually seeing the condition of the parts, a positive diagnosis under such circumstances is not possible, for the same symptoms may be due to a localised softening or cerebritis.

(*c.*) In others, again, the symptoms are suggestive of meningitis; in fact, in some cases the differential diagnosis of intracranial tumour and meningitis is very difficult.

Let us now consider *seriatim* the differential diagnosis of intracranial tumour, and of the various conditions with which it is likely to be confounded.

The Differential Diagnosis of Intracranial Tumour and Bright's Disease.

In those cases of Bright's disease in which there is little or no dropsy, headache and vomiting are often marked and distressing symptoms, the headache being in many instances confined to the back of the head. Now, in many cases of this description—and they are chiefly cases of chronic cirrhotic Bright—dimness of vision is a prominent symptom, and there are marked ophthalmoscopic appearances which it is not always easy, and which in some cases it is impossible, to distinguish from the ophthalmoscopic changes produced by intracranial tumours. In both conditions there may be epileptiform convulsions, or even genuine apoplectic seizures; and in both hemiplegia—a localising symptom, it will be noticed—may be present. Obviously, therefore, the two conditions may be easily mistaken the one for the other. The chief points to which attention should be directed in making the differential diagnosis are as follows:—

1. *The condition of the urine.*—The examination of the urine will generally clear up all difficulty and doubt; for in the majority of cases of chronic Bright's disease, which simulate intracranial tumour, distinct alterations indicative of the renal lesion—such as the presence of albumen and tube casts—are present in the urine.

It must, however, be remembered that in some cases of cirrhotic Bright—the form which is most likely to simulate intracranial tumour—albumen is scanty or even altogether absent; and that tube casts may be so few and far between as easily to escape detection. In cases of this description the amount of urine is considerably increased, and the specific gravity markedly below the normal.

2. *The condition of the heart, arteries, and blood pressure.*—In all forms of Bright's disease (except, in my opinion, pure and uncomplicated cases of waxy disease) the blood pressure is high, the arteries are apt to become atheromatous, and the left ventricle hypertrophied. These cardiac and vascular alterations are, however, much more marked in the cirrhotic form; indeed, in many cases, the degree of blood pressure and the amount of cardiac hypertrophy seem to be directly proportionate to the extent of the cirrhosis.

Fig. 43.—Sphygmographic tracing. High tension.

Obviously, therefore, the careful examination of the heart and arteries is a point of great diagnostic importance. A large hypertrophied left ventricle, without valvular disease to account for it, a hard pulse and characteristic sphygmographic tracing (see Fig. 43), or an atheromatous condition of the arterial walls, are strongly suggestive of chronic kidney disease, more especially cirrhosis.

3. *The exact character of the ophthalmoscopic changes.*—In the great majority of cases of intracranial tumour, the retinal change is a papillitis, of greater or less severity; and in the great majority of cases of renal disease the changes in the fundus oculi are those described under the term albuminuric retinitis (see Fig. 44). But in some cases of tumour the retinal changes resemble, more or less closely, those of Bright's disease, white spots or streaks being present in the neighbourhood of the macula; and *vice versâ*, in some cases of Bright's disease, the white patches are absent and the retinal change is essentially a papillitis.

Again, the appearance in the fundus (both in tumour and Bright's disease) may be suggestive of either, but characteristic of neither affection.

To sum up, in most cases it is easy to decide, by means of the ophthalmoscope, whether the retinal changes are characteristic of cerebral tumour or of kidney disease; but in some cases this is

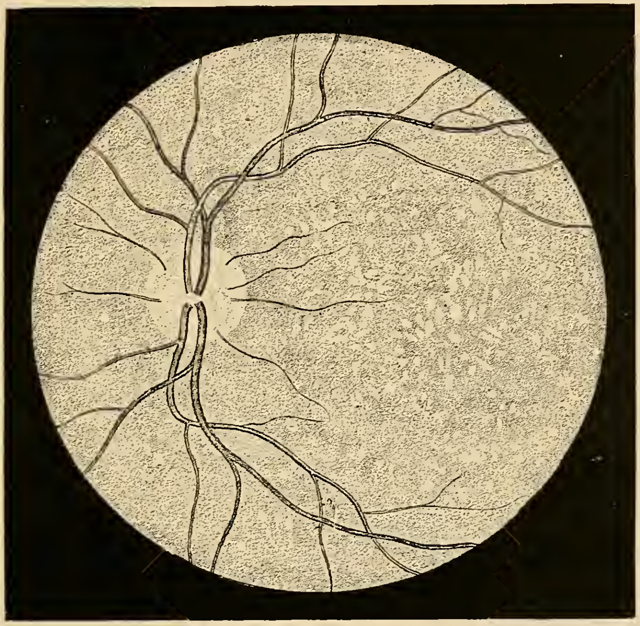

Fig. 44.—Fundus oculi in a case of albuminuric retinitis. Numerous white patches are seen in the retina, more especially in the neighbourhood of the macula.

difficult or impossible. The ophthalmoscopic appearances, *per se,* can never be taken as an absolutely certain guide, for the appearances typical of one condition are, in some rare and exceptional cases, met with in the other.

In reference to this point, Dr. Gowers says—" Although the resemblance in the appearance of the fundus in the two cases is sometimes close, it is rarely that the diagnosis cannot be made by a careful examination. The white spots about the macula are the result of damage produced during the acute stage of the neuritis, when it involves the adjacent retina; and, as an almost invariable rule, when

they are seen as scattered spots resembling those of Bright's disease, the inflammation of the papilla is distinctly subsiding and the swelling has become pale, although its characters (constriction of vessels, &c.) indicate that the previous inflammation was great in degree. On the other hand, in most cases of Bright's disease in which the scattered white spots appear, the papilla is but little affected by inflammation, and that usually shows no signs of subsidence. Considerable difficulty only exists in the cases just spoken of, in which the nephritic retinal changes are accompanied by a preponderating neuritis, which runs its course and then subsides. During the stage of subsidence the appearance of the fundus may be exactly similar to that in some cases of cerebral disease or of primary papillitis."[1]

4. *The character of the headache.*—In Bright's disease the headache is not as a rule so severe and unbearable as it is in many cases of tumour. Very severe and distressing headache is therefore suggestive of tumour. The contrary proposition (that mild headache is suggestive of Bright's disease) does not, however, hold good; for in some cases of tumour the headache is, as we have seen, slight.

In many cases of Bright's disease the headache is chiefly occipital; but this fact is of little value for diagnostic purposes, for in some cases of tumour (more especially when the new growth is situated beneath the tentorium) the same localisation of the headache is observed.

5. *The age of the patient.*—This may be of some importance, for cirrhotic Bright's disease (the form most likely to be confounded with tumour) rarely affects young people. The fact that the patient is young (under twenty years) is therefore in favour of tumour. Absolute reliance cannot, however, be placed on this, nor indeed upon any other single fact or symptom.

The important lesson to be drawn from the foregoing statements (which show the close resemblance that there sometimes is between the symptoms produced by an intracranial tumour and Bright's disease) is *never* to commit oneself to a definite diagnosis of intracranial tumour, *without having previously examined the urine and excluded Bright's disease.*

It must also be remembered that in rare cases the two diseases (Bright's disease and intracranial tumour) are combined. Hence the

[1] *Transactions of the Ophthalmological Society of the United Kingdom*, vol. i., p. 103.

fact that there is Bright's disease (as shown by the condition of the urine, &c.) does not absolutely prove that there is no tumour. The numerical chances are strongly against such a combination, and in the great majority of cases it would be quite safe, having detected kidney disease, to stop there and to exclude tumour. In a few cases such a method of diagnosis would lead to error, and the physician should always ask himself (having detected Bright's disease), whether the kidney lesion is sufficient to account for the cerebral symptoms.

Dr. Hughlings Jackson says, with reference to this point—"I have seen double optic neuritis in a man who had unilaterally and deliberately beginning convulsive seizures—a condition pointing mostly, in such association, to cortical tumour—and Bright's disease too. I saw a case, many years ago, under the care of Dr. Habershon, in which there were found, post mortem, both renal disease and intracranial tumour, although in this case intracranial disease was diagnosed from other nervous symptoms." [1]

The Differential Diagnosis of Intracranial Tumour and Lead Encephalopathy.

In the slighter forms of lead poisoning, such as are usually met with in private practice, head symptoms are rarely observed; but when large quantities of lead are introduced into the system, very grave symptoms, which in some cases closely resemble those produced by intracranial tumours, may arise. Cases of this kind not infrequently prove fatal.

Persons employed in the manufacture of white lead often suffer in this way; and in Newcastle such cases used frequently to come under my notice.

The symptoms characteristic of lead encephalopathy may commence gradually, or with more or less suddenness. They usually consist of severe headache, vomiting, and convulsions, the spasms being in some cases epileptiform, in others of the hystero-epileptic type. On ophthalmoscopic examination intense double optic neuritis is usually, but not invariably present. In *Brain*, January 1888, page 507, I have recorded a case of lead poisoning in which marked

[1] *Transactions of the Ophthalmological Society of the United Kingdom*, vol. i., p. 62.

head symptoms and amaurosis were present, but in which the discs were absolutely healthy. The pupils are usually dilated, sluggish, or absolutely insensible in their reaction to light. In some cases there is strabismus; in the few cases in which I have observed this symptom it was double and internal. During the acute stage there is frequently a condition of mental aberration and excitement, in which the patient may be so violent as to require restraint. This condition may last for some weeks. In other cases—and these are usually the most severe, and not infrequently fatal—coma, rather than maniacal excitement, alternates with the convulsions. In fatal cases the patient may be profoundly comatose for many hours before death.

The symptoms, more especially after the acute stage has passed off, may closely resemble those due to an intracranial tumour; and in those cases in which no previous history is forthcoming, the difficulties of diagnosis to the great mass of medical men, who have had no opportunity of seeing cases of lead encephalopathy, may be considerable. So closely do the two conditions (tumour and lead encephalopathy) in some cases resemble one another, that I never commit myself to a positive diagnosis of intracranial tumour *without having previously excluded lead poisoning.*

The differential diagnosis is easily enough made, if one is on one's guard, by attention to the following points.

1. *The presence of a blue line on the gums, and other symptoms of lead poisoning, such as anœmia, colic, constipation, wrist drop.* The characteristic blue line on the gums is almost always present. It may be absent if the patient has no teeth, or if the teeth are kept scrupulously clean, for it is due to the action of sulphuretted hydrogen (produced by the decomposition of food and other matters lodging around the roots of the teeth) on the lead, which is circulating through the tissues. The class of people employed in the more dangerous parts of the process of manufacturing white lead are, in my experience, essentially dirty; they are, too, usually *young* women, and therefore have teeth. Hence the blue line is almost always well marked. In one of my cases, although the patient had not been exposed to the poison for fourteen months, the blue line was still quite perceptible. Dr. Richardson (quoted by Hamilton in his work on the diseases of the nervous system, page 472) states that in

America the men suffer more frequently from lead encephalopathy than the women.

Since writing the above I have met with a case of lead encephalopathy in which the blue line was practically absent: in it there was only one small patch of blue deposit in a spongy and unhealthy portion of the gum. The patient, who knew the risk he ran of lead poisoning, had been scrupulously careful to keep his teeth clean, and to brush them regularly on returning from work.

2. *The previous history.*—In most cases of lead encephalopathy the patient has, prior to the commencement of the present illness, suffered from the ordinary well-known symptoms of lead poisoning, such as colic, wrist drop, lead rheumatism, &c.

3. *The occupation of the patient* is a point of importance, for, as far as I know, the severe cerebral symptoms to which the term lead encephalopathy is given do not occur in plumbers, painters, and others who so commonly suffer from the milder manifestation of lead poisoning, but, in this country at all events, are usually confined to persons employed in the manufacture of lead compounds.

4. *The character of the convulsions and the peculiar mental condition,* which have been described above, are also strongly suggestive of lead poisoning rather than of cerebral tumour.

It would be out of place to discuss here the nature of the pathological changes which are the cause of lead encephalopathy. Suffice it to say that in some cases the symptoms are due to the action of the poison upon the nervous tissues themselves (brain, optic nerve, &c.); in others kidney disease and resulting uræmia seem to play an important part in their production. It is probable that in many cases the cerebral symptoms are due to a combination of these lesions. In all cases of suspected lead encephalopathy the urine should be carefully examined, and the exact condition of the kidneys determined.

5. *The presence of lead in the urine.*—On chemical examination lead can probably be detected in most cases in the urine. In the case of lead amaurosis without neuritis, which has been previously referred to, Dr. Stevenson Macadam, who kindly examined one specimen for me, failed to detect the presence of lead in the urine.

The Differential Diagnosis of Hypermetropia, with or without Anæmia, and of Intracranial Tumour.

Young persons affected with errors of refraction not infrequently suffer from distressing headache ; and Mr. Cowper states that the prolonged use of the eyes in some cases of hypermetropia actually induces œdema and swelling of the disc, which is occasionally mistaken for, and treated as, slight optic neuritis depending upon cerebral disease.

Dr. Gowers has recorded several cases of optic neuritis in girls suffering from chlorosis and hypermetropia, in which the anæmia seemed to be the most effective cause of the optic neuritis, for under the administration of iron the sight became normal, although the eyes were used and the hypermetropia was uncorrected.

It would appear, therefore, that both anæmia and hypermetropia are in some cases the cause of optic neuritis, and that the tendency to inflammation of the optic papilla is intensified when both causes exist in combination.

In cases of this description the differential diagnosis is, as a rule, easily made.

The headache is not usually so severe as it is in well-marked cases of intracranial tumour ; it is chiefly orbital or frontal ; is increased by using the eyes, more especially by all efforts which require active accommodation.

The fact that there is an error of refraction ; that on correction of that error by suitable glasses, or by disuse of the eyes, the headache is relieved ; the circumstance that light hurts the eyes and aggravates the symptoms ; the slight degree of the optic neuritis, and its subsidence under treatment ; together with the absence of any localising symptoms, are usually sufficient to clear up all doubts as to the nature of the case.

There are, however, some cases of anæmia, in which the symptoms of intracranial tumour are so closely simulated, that a differential diagnosis is very difficult or altogether impossible. The most striking cases of this kind with which I am acquainted were related by Dr. Broadbent at the great debate on the causation of double optic neuritis which took place at the Ophthalmological Society a few years ago.

Dr. Broadbent stated on that occasion "that he had recently had under his care at St. Mary's Hospital a young girl who for two years had had the usual symptoms of cerebral tumour—very severe pain in the head, vomiting, and double optic neuritis. She was quite blind from atrophy of the discs, evidently consecutive upon neuritis; she still had, when admitted, violent paroxysmal headache and vomiting, and passed her urine and fæces in bed, not from unconsciousness but from indifference. It was ascertained that the symptoms set in immediately after sudden suppression of the catamenia, and that she had never menstruated during the two years of her illness. The treatment, therefore, was directed to the restoration of this function, and the flux soon appeared. From this time the headaches ceased; she became cleanly, and, except for the blindness, was well."

In another case, in which headache, vomiting, and double optic neuritis had been coincident with sudden arrest of the menses, there had been a fatal termination after many fluctuations, and he had had the opportunity of making a post-mortem examination. There was no tumour nor meningitis, and the only morbid appearance in the brain was effusion into the ventricles.[1]

The Differential Diagnosis of Atrophy of the Brain and Intracranial Tumour.

In some very rare and quite exceptional cases, in which the characteristic symptoms of an intracranial tumour (headache, vomiting, and double optic neuritis) were present during life, no coarse lesion of the brain, but only sclerosis, atrophy, or microscopical lesions, have been found at the post mortem.

In such cases a correct diagnosis is obviously impossible.

These cases show that the general symptoms of intracranial tumour are not in all cases sufficient for a correct diagnosis. Unless localising symptoms are at the same time present, the most experienced observers will occasionally, in the present state of our knowledge, make mistakes, and fail to find on post-mortem examination the tumour which had been confidently expected during life. It remains to be seen whether more extended observation and experience will enable a distinction to be made in cases of this description.

[1] *Transactions of the Ophthalmological Society of the United Kingdom*, vol. i., p. 108.

A remarkable case, illustrative of the foregoing statements, has been reported by Dr. Stephen Mackenzie.[1]

The Differential Diagnosis of Intracranial Tumour and Migraine.

In typical cases of ordinary migraine the distinction is easily enough made, but in irregular forms there may be great difficulty in arriving at a positive conclusion. Further, it must be remembered that a patient, who has for years been subject to ordinary migraine, may subsequently be affected with an intracranial tumour. In cases of this kind, unless the symptoms are very clearly marked, the tumour may easily be overlooked. This is particularly apt to be the case when there is no optic neuritis.

The markedly periodic character of the attacks of headache and vomiting; the exact character of the pain and its localisation; the presence of temporary hemianopsia, fortification vision, &c., at the onset or during the attack; the protracted course of the case; and the fact that between the paroxysms there are no indications of intracranial mischief, are strongly in favour of migraine—a condition which is not infrequently hereditary. The diagnosis of migraine is strengthened or confirmed by the absence of distinct evidence of organic disease—in particular, by the absence of double optic neuritis and of localising symptoms; and in some cases by the effects of treatment. (Some cases of migraine speedily yield to guaranina or antifebrin given at the commencement of the attack, and to the systematic administration of arsenic or other nerve tonics during the intervals.)

In those cases in which symptoms of migraine are associated with hysteria or great general nervousness, the difficulty of diagnosis is much increased.

Further, it must be remembered that in some cases of tumour the headache closely resembles that of ordinary migraine; indeed, no less an authority than the late Dr. Hilton Fagge supposed (as has been stated on page 27) that the headache of tumour and of migraine are produced in exactly the same manner.

[1] *Brain*, vol. vii., p. 257.

The Differential Diagnosis of Intracranial Tumour and Hysteria.

Hysterical symptoms are not infrequently associated with intracranial tumour, and indeed with all forms of organic disease; and in some cases, in which the symptoms and signs of hysteria are prominent and the symptoms and signs of the organic lesion (tumour), with which the hysteria is associated, are ill developed, the differential diagnosis may be a matter of extreme difficulty; in some cases it may be impossible to come to a positive conclusion.

The diagnosis of an intracranial tumour in a person of highly neurotic temperament is, in the absence of optic neuritis and positive evidence of localised organic disease, extremely difficult. In cases of this description it is very difficult to decide what value should be attached to headache and other subjective symptoms.

In all doubtful cases, therefore, the observer must endeavour to make himself familiar with the original (normal) mental constitution of the patient, in order that he may be able to attach the true value to her symptoms and complaints.

In dealing with persons who are highly neurotic or distinctly hysterical, it is essential to remember that the diagnosis of mere hysteria (nothing more than hysteria) is never justified, unless the observer has, after repeated examinations, failed to detect symptoms or signs indicative of organic disease.

The condition of the optic discs is without doubt the most important point in clearing up the diagnosis. The presence of double optic neuritis enables the observer to decide in favour of tumour, for this symptom is very rarely, if ever, associated with mere hysteria alone. It will be observed that I do not say double optic neuritis is *never* associated with mere hysteria. For not only would such a dogmatic method of statement be, in my opinion, highly unscientific, but I am personally disposed to think that it would, as a matter of fact, in this case be erroneous, or apparently erroneous. Double optic neuritis, as we have seen, *is* occasionally met with in anæmia, and in association with disordered menstruation, with errors of refraction, and with some other conditions, such as diffused cerebritis, in which there is no "coarse" intracranial lesion, and with which hysterical symptoms are not infrequently associated. Now in such cases the hysterical

condition may easily appear to be the only cause for the ocular change. Further, we are still in doubt as to the exact pathology of hysteria and hysterical symptoms (hysterical paralysis, hysterical contracture, hysterical anæsthesia) on the one hand, and of the double optic neuritis associated with cerebral tumours on the other. In the presence of such ignorance, it would, in my opinion, be rash and unscientific to affirm that the same (?) functional alterations which produce hysterical paralysis, hysterical contracture, or hysterical anæsthesia, *cannot* possibly lead to the production of some swelling of the optic papilla. My own view is that papillitis is not at all likely to be produced in this way; but, as has been previously stated, Dr. Hughlings Jackson and some other eminent authorities consider the vaso-motor theory as to the mode of production of optic neuritis the best that has yet been advanced. And if it is granted that organic disease can produce papillitis in this way, it must perhaps be allowed that the functional alterations in the nerve centres, which are the cause of hysteria, may also produce vascular changes (œdema and inflammation) in the optic discs.

Personally I am disposed to think that the functional changes in the nerve centres, which produce hysterical paralysis or hysterical contracture, are not in the least likely to lead to the production of double optic neuritis; but while holding this view, I cannot, for the reasons given above, assert dogmatically that double optic neuritis is *never* associated with mere hysteria alone, or that it *cannot possibly* be produced in this manner.[1]

But be this as it may, double optic neuritis is so rarely, if ever, met with in connection with mere hysteria, and so commonly associated with intracranial tumours, that its presence (in a case in which the diagnosis lies between hysteria and cerebral tumour) practically settles the question, and, in the present state of our knowledge, may be taken as a definite indication that the case is not one of mere hysteria alone.

[1] The experience of any single observer is necessarily so limited, and the knowledge which can be acquired by making oneself familiar with the accumulated experience of authorities, is, when compared with the facts and possibilities of nature, so imperfect, that I question whether such terms as *never*, *cannot*, and the like should ever be used in an unlimited and unqualified sense. It is of course quite another thing to use these terms in a limited and qualified manner, and to say, for instance, *so far as I know*, or *so far as is known*, such and such symptoms *never* or *cannot* occur in such and such conditions

But while the ophthalmoscope enables us to clear up the diagnosis in those cases in which double optic neuritis is present, it does not of course enable us to make a diagnosis in all.

In those cases in which there is no double optic neuritis, the severity of the headache is a point of some importance; but in hysterical and neurotic patients it is often (as has been already pointed out) a difficult matter to gauge the severity of the headache and other subjective symptoms. The purposeless vomiting, too, which is so highly suggestive of organic cerebral disease, may be exactly simulated by the vomiting of hysteria; while the convulsive seizures of hysteria and of organic disease are sometimes difficult to differentiate, more especially when they are not seen by the physician himself. Again, in hysteria, paralyses (which in some cases are most difficult to distinguish from the paralyses due to organic disease) may be present.

Localised epileptiform convulsions, or a distinct epileptic fit, are strongly suggestive of organic disease. Localised paralysis of the muscles supplied by a cranial nerve (though not very common in cases of tumour) is, when present, of the greatest diagnostic value; for, while paralysis of the limbs is not uncommon in hysteria, paralysis of the face is rarely observed even in hysterical hemiplegia; and isolated paralysis of muscles supplied by motor cranial nerves, such as the facial, is very rarely due to hysteria alone.

In doubtful cases, a careful study of the distribution of the paralysis, of the state of the reflexes, and indeed of the condition of the whole nervous system, together with a judicial survey of the previous history, whole progress, and course of the case, will generally enable the physician to come to a positive conclusion as to whether the case is merely one of hysteria alone, or whether, in addition to the hysterical symptoms or general neurotic (nervous) condition, there is not some associated *organic* disease (*e.g.*, tumour).

In all doubtful cases, a history of the previous removal of a tumour, the fact that there is a new growth in some other organ, or the presence of a constitutional state, which is likely to predispose to the production of certain forms of new growths (such as the syphilitic and scrofulous), should be diligently inquired for.

The Differential Diagnosis of Intracranial Tumour and some forms of Insanity.

Provided that the physician is acquainted with the previous history and course of the case, there is seldom any difficulty in distinguishing these conditions.

In the later stages of those cases of tumour in which the mental deterioration is extreme, the patient may make no complaints, and the symptoms of "coarse" lesion may be so little marked as to pass unnoticed. It is obvious that under such circumstances the condition may be mistaken for ordinary dementia (*i.e.*, dementia without "coarse" organic disease), and an erroneous diagnosis arrived at, should the physician be unacquainted with the earlier symptoms of the case.

Again, in those rare cases of intracranial tumour in which maniacal symptoms are developed, unless the previous history and course of the case are known to the physician, the presence of a tumour may be unsuspected.

If Lautenbach's observations, which show that well-marked double optic neuritis is present in about 16 per cent of cases of acute mania, are confirmed, the distinction of acute mania with "coarse" organic disease, from acute mania without "coarse" organic disease, will, in the absence of a definite history of previous symptoms of organic lesion (severe headache, vomiting, and localising symptoms), be practically impossible.

The Differential Diagnosis of Intracranial Tumour and of Meningitis.

This is in most cases easy, in some most difficult, and in others impossible. In many cases, indeed, the two conditions are combined.

In some cases it is impossible to draw a distinction during life between a syphilitic cerebral tumour (gumma) and syphilitic meningitis, or between a tubercular tumour and a localised tubercular meningitis, more especially of the convexity.

The diagnosis is especially difficult in those cases of meningitis (and they are usually tubercular or syphilitic) which run a protracted (subacute or chronic) course, on the one hand; and in those cases of

cerebral tumour, on the other, which rapidly develop, which follow injury, and which are associated with meningitis or cerebritis.

In both conditions (tumour and meningitis) headache, vomiting, double optic neuritis, and general or localised convulsive seizures, may be prominent symptoms; and in some cases of meningitis there may even be localised paralysis.

The cases in which a distinction can usually be made are cases of cerebral tumour on the one hand, and *generalised* meningitis on the other.

The chief points to which attention should be directed in order to differentiate the two conditions are—

1. *The mode of onset, course, and duration of the case.*—In ordinary typical cases of tubercular meningitis, such as occur in children, the onset is as a rule much more sudden; the symptoms advance much more steadily and progressively from bad to worse, a distinct stage of irritation usually preceding a stage of depression; and the whole duration of the case, averaging as it does about two weeks, is much shorter than that of most cases of intracranial tumour.

In quite exceptional cases tubercular meningitis runs a much more protracted course. The diagnosis is then much more difficult. I have myself reported a case of meningitis (I believe from the post-mortem evidence tubercular) in which the inflammatory symptoms gradually subsided and the patient died six months after the onset of the attack, from hydrocephalus, secondary to the meningeal inflammation. A duration of more than six weeks is strongly against meningitis. With regard to this point the late Dr. Hilton Fagge says, "Now I believe it may be said that wherever cerebral symptoms of the kind described in this chapter (*i.e.*, symptoms characteristic of an intracranial tumour) have lasted for more than five or six weeks, the only other organic affections of the brain by which they can be caused are abscess and certain diffused morbid changes."[1]

While personally I am not prepared to endorse to the full this statement, I nevertheless admit the great diagnostic importance of a protracted course (*i.e.*, of a duration of more than six weeks).

A careful inquiry into the exact mode of onset of the attack and the previous condition of the patient is of great importance; for cases

[1] *Principles and Practice of Medicine*, vol. i., p. 539.

of intracranial tumour are sometimes met with, in which the onset appears to be acute, and attended with febrile symptoms. In almost all cases of this kind, which are very liable to be mistaken for cases of meningitis, a careful inquiry will usually, I believe, elicit a history of previous cerebral symptoms (headache, vomiting, slight mental alterations, &c.). In the great majority of cases of this kind the tumour has been slowly developing (without, perhaps, producing any marked cerebral disturbance) for some time before the apparent acute onset, which is usually due to the occurrence of meningitis, cerebritis, or some other acute change in the neighbourhood of the new growth.

In very rare cases a patient who is already the subject of commencing cerebral disease (tumour) receives a head injury, which is followed by well-marked symptoms of meningitis. Unless the condition of the patient prior to the receipt of the injury is carefully elicited, it is quite obvious that the meningitis may, under such circumstances, be considered the primary lesion, and the previous disease (tumour) overlooked. I have known a case of this kind in which a patient, already the subject of a cerebral tumour, fell (apparently in consequence of vertigo or an epileptic fit) and severely injured his head, the injury being followed by well-marked cerebral symptoms.

In cerebro-spinal meningitis the onset is, as a rule, even more sudden, the symptoms more rapidly progressive, and the duration shorter than in tubercular meningitis. There is not much likelihood of confounding cases of this kind with cases of intracranial tumour.

In some of the cases in which the onset is more or less sudden, and symptoms common both to meningitis or tumour (headache, vomiting, optic neuritis, vertigo, &c.) are developed, the headache and other symptoms disappear, the optic neuritis completely subsides, and the recovery may be perfect. In cases of this kind I am disposed to think that the lesion was more probably meningitis, often syphilitic meningitis, rather than tumour. It must, however, be admitted that the lesion may have been a syphilitic gumma; but other tumours may, I think, be excluded, for all pathologists know that nothing is more rare than to find the remains of a cured tumour (other than a syphilitic gumma) within the skull; and if the cases I am now referring to were due to a "coarse" lesion (tumour), the remains of cured tumours ought occasionally to be found.

Some of our best authorities differ from this opinion, and think that in such cases the pathological condition was tumour (" coarse " disease) rather than meningitis. Dr. Hughlings Jackson, for example, says—" Suppose next that there was an acute illness from which the patient has recovered, except for defective sight, with some atrophy of the optic discs. Here comes the question, was the illness in either this or the former case meningitis ? For my own part, I believe it to be more probable that it was, and remains a case of local, gross organic disease, such as tumour. It is of no avail to say of the acute case that the patient had all the symptoms of a meningitis, for there are no symptoms known to be characteristic of meningitis only. Admitting that during the illness we might be unable to decide, I should take the mere fact of recovery as strong evidence against meningitis ; not that I need deny that recovery from meningitis occasionally happens. Virchow speaks of recovery from tubercular meningitis. Autopsies alone can decide. So far as autopsies go (made some time after recovery on return of head symptoms, or after death by other cause), the evidence is in favour of local, gross organic disease, rather than of meningitis in cases of the kind referred to. I feel sure, at any rate, that facts show that even complete recovery, with good sight, does not negative persisting local, gross organic disease within the cranium. This is strikingly true of some cases of syphilitic tumour; the symptoms pass off under treatment, but the patient may come again and again for the same localising symptom (for convulsions beginning in one hand, let me instance), showing disease persisting in one place, as in a striking case (of which I am thinking) the necropsy proved. So, instead of asking for evidence from cases of patients who have recovered from symptoms of meningitis, I ask the *question*, Have traces of meningitis been found after such recoveries, when patients have died later, or of some other disease ?"[1]

2. *The behaviour of the temperature and pulse.*—Elevation of temperature, and increased frequency of the pulse in the early stages, followed by slowing and irregularity in the after periods, are in favour of meningitis rather than of tumour. When the tumour is complicated with meningitis or cerebritis, there may of course be some febrile disturbance, but it is seldom prominent.

[1] *Transactions of the Ophthalmological Society of the United Kingdom,* vol. i., p. 73.

3. *The severity and character of the headache.*—These are sometimes of importance. Very severe and localised headache is certainly in favour of tumour. In meningitis there is usually, I think, more hypersensitiveness to light, noise, &c., than in tumour.

4. *The degree of optic neuritis, and, in tubercular cases, the presence of tubercles in the choroid.*—In rapid cases of tubercular and other forms of meningitis, optic neuritis is seldom present, but in the more protracted cases it is often observed. In tubercular cases the degree or intensity of the neuritis is seldom so great as in cases of tumour. The intensity of the neuritis is not, however, an absolutely safe guide, for in cerebral tumours the neuritis may of course be slight, while in some cases of meningitis[1] (tubercular and other) the most intense neuritis, followed by optic atrophy, is observed. "Apart from the distinguishing characters afforded by the degree of the neuritis," Dr. Gowers states that he has "noted only one difference between the neuritis caused by tumour and that due to meningitis, viz., that during the early stage of inflammation, when it is coming on and the swelling is distinct, the papilla is paler in meningitis than in tumour."[2]

Dr. Hughlings Jackson states that "in tubercular meningitis the swelling is slight, not extensive; it is even, merges into the fundus; the disc is succulent-looking; there is not time possibly for further development. It is like the earliest stage of optic neuritis from intracranial tumour."[3]

In long-continued cases of tubercular meningitis, tubercles are not infrequently found in the choroid on post-mortem examination; their detection during life, while it would undoubtedly show the pathological nature of the lesion, and would be in favour of meningitis rather than tumour, would not necessarily exclude a scrofulous tumour, with which tubercles in the choroid may of course be associated.

In two cases in which numerous large white spots were present in the neighbourhood of the macula, I had considerable difficulty in deciding whether they were in the retina and due to Bright's disease, or whether they were tubercles in the choroid—a difficulty which in one of the cases was also shared by an experienced observer.

[1] See a case reported by me in the *Edinburgh Medical Journal* for August 1879.
[2] *Transactions of the Ophthalmological Society of the United Kingdom*, vol. i., p. 104.
[3] *Ibid.*, p. 64.

The presence, then, of tubercles in the choroid is in favour of meningitis, but does not exclude tumour.

5. *The age of the patient.*—This is of some importance, for in young children tubercular meningitis is more common than tubercular tumour, while in adults the reverse is the case.

The Differential Diagnosis of Intracranial Tumour and of Cerebral or Cerebellar Abscess.

In some cases the distinction is extremely difficult, indeed it may be impossible. An encapsuled abscess must in fact be looked upon as a (cystic) tumour, and may be associated with the ordinary symptoms of a new growth—headache, vomiting, optic neuritis, vertigo, &c.

In order to differentiate the two conditions, attention should be chiefly directed to the following points:—

1. *The presence or absence of a cause of abscess.*—Cerebral abscesses very rarely develop *per se*, but usually have their starting-point in suppurative disease of the ear, nose, or some other part of the cranial bones. In some cases abscess is secondary to an injury of the head. In others a lesion within the thorax, more especially lung disease, is the source of origin.

In other cases—but these I do not include in speaking of localised cerebral abscess—the cerebral lesion is part and parcel of a general pyæmia; in cases of this kind the abscesses are almost always multiple, usually of small size, and generally associated with diffuse meningitis or cerebritis, or both.

In all cases, therefore, in which there is any doubt as to the diagnosis, the condition of the ear, nose, and other parts of the skull should be carefully examined; in fact, I never commit myself to a positive diagnosis of intracranial tumour, *unless I have excluded suppurative ear and nose disease.*

In those cases in which a local cause of abscess (such as suppurative ear disease) *is* present, in addition to the general symptoms suggestive of tumour or abscess (headache, vomiting, and it may be double optic neuritis), the chances are strongly in favour of abscess rather than of tumour. It is of course possible to have an intracranial tumour and an otorrhœa (or other local cause of cerebral abscess) accidentally associated, but the chances are strongly against

such an accidental occurrence. *Vice versâ,* when no discoverable local cause (source) of abscess is present, the chances are, other things being equal, very strongly in favour of tumour, and the correct diagnosis of cerebral abscess in such circumstances (*i.e.*, in the absence of disease of the ear, nose, or cranial bones, and of traumatic injury) is seldom more than a fortunate guess.

2. *The previous history, whole progress, and course of the case.*—This may throw light on doubtful cases; when no clear or reliable evidence is forthcoming the difficulties of diagnosis are much increased.

A history of former ear disease, with the subsequent long continuance of cerebral symptoms, is in favour of abscess. The rapidity with which the symptoms progress does not afford any certain guide, for in some cases of encapsular abscess (*i.e.*, while the abscess is still encapsuled and unassociated with cerebritis, meningitis, or sinus phlebitis) the course of the cases is very chronic, and there may be few, if any, characteristic symptoms. In tumour, the same absence of striking symptoms may also of course be observed. In tumour, headache is, I think, usually more severe. This statement, as the former, only applies to encapsuled abscesses (while still encapsuled), for I have known the most intense headaches which it is possible to conceive in cerebral abscess. In the case to which I refer there were three separate abscesses (one in the frontal, one in the temporo-sphenoidal lobe, and one in the cerebellum).

Double optic neuritis is also, I think, more frequent in tumour. Possibly unilateral optic neuritis may be in favour of abscess; but more information is required before it is possible to generalise on this point.

The temperature may afford no guide; for in encapsuled abscess, as in tumour, it is usually normal or subnormal. A suppurative temperature, with rigors and sweatings, is of course strongly in favour of abscess. Emaciation without sufficient apparent cause is perhaps suggestive of abscess.

In a doubtful case, the *sudden* occurrence of symptoms indicative of severe, diffuse meningitis would be strongly in favour of abscess. Symptoms and signs of sinus phlebitis also, of course, point strongly to abscess.

The situation of the lesion is a matter of some importance; for

abscesses are in the great majority of cases situated either in the temporo-sphenoidal lobe or the cerebellum ; the location of the lesion in some other part of the brain is therefore in favour of tumour.

The Differential Diagnosis of Extra- and Intracranial Syphilis.

In some cases of extracranial syphilis, in which the headache is severe, the tenderness of the scalp great, the periosteal thickening evident, and the patient exhausted by sleeplessness and suffering, both the patient and the physician may suspect the presence of an *intra*-cranial lesion. The suspicion is not unnatural ; for intracranial nodes or other intracranial syphilitic lesions are frequently combined with extracranial syphilis. Difficulties in diagnosis are especially apt to occur in those cases in which a periosteal lesion, surrounding the orifice of exit of a cranial nerve, produces well-marked and localised paralysis or anæsthesia. The points to which attention is chiefly to be directed in making the differential diagnosis are—

1. The condition of the optic discs.

2. The presence or absence of definite cerebral symptoms, such as vomiting, vertigo, paralysis, or anæsthesia of cerebral distribution, spasms or convulsive twitchings, hemianopsia, &c.

3. The therapeutic effects of a vigorous anti-syphilitic treatment. The fact that as the external nodes disappear the headache and other symptoms entirely subside, does not of course prove that the lesion is entirely *extra*cranial ; for under the influence of large doses of iodide of potassium (at least a drachm and a half daily) internal syphilitic lesions often clear up with great rapidity. But the fact that, with the disappearance of the external manifestations, the headache and other cerebral symptoms entirely subside, is very suggestive that the lesion is extracranial only ; for the meningitic and vascular (cerebral) lesions seldom clear up with the same rapidity as the periostitis.

The Differential Diagnosis of Cerebral Tumour and Cerebral Hæmorrhage.

The distinction is usually easy, provided that the previous history of the case is accurately known.

The strong probability is, of course, that an apoplectic attack in

a patient (previously unknown and about whom no history is forthcoming) is due to ordinary cerebral hæmorrhage, rather than to a hæmorrhage or to a pseudo-apoplectic attack associated with tumour. The physician who aims at being as correct as possible in every case, will not, however, rest satisfied with mere probabilities, but will try to make an exactly accurate diagnosis in every case. And there can be little doubt that hæmorrhages from vascular gliomata have not infrequently been mistaken both during life and after death for ordinary hæmorrhagic apoplexies.

The chief points to which attention should be directed in order to distinguish the conditions are—

1. *The history of the case and mode of onset of the attack.*—In those cases in which the symptoms of cerebral tumour are well marked, and the history of the case is known to the physician, there is little or no difficulty.

When an apoplectic attack has been preceded by attacks of headache, vomiting, &c., the possibility of intracranial tumour should always be kept in view. (Bright's disease may, as has been previously pointed out, produce the same symptoms.)

2. *The condition of the optic discs.*—The presence of double optic neuritis in a case of supposed apoplexy (ordinary cerebral hæmorrhage) is strongly suggestive of the presence of a tumour (more especially if there is no renal disease and no plumbism), for cerebral hæmorrhage *per se* is seldom, if ever, attended with double optic neuritis.

3. *The age of the patient.*—Ordinary cerebral hæmorrhage is rare before middle age, while the cerebral hæmorrhage or pseudo-apoplectic attacks connected with tumour may occur at any age, and are just as frequent, perhaps even more frequent, in young than in old people.

4. *The condition of the urine.*—This is most important, for in many cases of ordinary cerebral hæmorrhage the kidneys are cirrhotic.

5. *The presence of associated lesions,* such as are to be met with in cases of tumour on the one hand (syphilitic or scrofulous lesions, sarcomatous and cancerous deposits in other organs), and of ordinary cerebral hæmorrhage on the other (albuminuria, hypertrophy of the left ventricle without valvular disease, high arterial tension, albuminuric retinitis, atheroma of the peripheral vessels, &c.).

The differential diagnosis of the pseudo-apoplectic attacks, which are so common in some forms of tumour, more especially in cases of cerebral syphilis, and *of apoplectic attacks due to hæmorrhagic extravasations,* such as occur so frequently in cases of glioma, is a point of some importance; for the prognosis is markedly different in the two conditions,—pseudo-apoplectic attacks are usually recovered from, while apoplectic seizures, due to large hæmorrhagic extravasations from the vessels of a tumour, are often fatal.

The distinction cannot always be made with certainty; the chief points to which attention is to be directed are—

1. *The nature of the tumour.*—As stated above, hæmorrhagic extravasations are chiefly met with in connection with glioma; but they may occur in cases of sarcoma and cancer,—in short, in all cases in which there is excessive vascularity either in or around a new growth. Pseudo-apoplectic attacks, though probably more common in syphilitic than in other forms of new growth, are by no means distinctive of that condition. They may occur in cases of glioma, sarcoma, cancer, &c.

2. *The behaviour of the temperature.*—This is in many cases a point of considerable diagnostic value; for in pseudo-apoplectic seizures the temperature alterations which occur in cases of large cerebral hæmorrhage are not usually observed.

3. *The progress of the case.*—In pseudo-apoplectic attacks, the unconsciousness, hemiplegia, &c. soon pass off (provided, of course, that the patient recovers from the coma), while in genuine apoplectic seizures the unconsciousness is often prolonged, the result much more frequently fatal, and, if the patient does recover, the paralysis is much more apt to remain.

These facts are of importance as regards prognosis, for in cerebral syphilis the prognosis is never absolutely hopeless, even although the unconsciousness is profound, and the symptoms apparently most desperate.

CHAPTER VIII.

DIAGNOSIS—*continued:* THE LOCALISATION OF THE TUMOUR.

Having decided that there is an intracranial tumour, we endeavour in the next place to determine its exact locality or site—a point of the greatest importance, both for practical (operative) and scientific purposes, for it is only by observing the effects of localised brain lesions, and, as Horsley has shown, by electrical irritation of portions of the cortex exposed during surgical operations, that we can, *in man*, determine the function of special parts of the brain tissue.

The local diagnosis is arrived at by observing—

1. The exact nature of the *positive* localising symptoms, which show derangement of function in special parts of the brain tissue.

2. The *negative* indications, or absence of symptoms indicative of functional derangement of special parts.

3. The manner in which the symptoms (positive and negative) are grouped together, and the order in which they are developed.

Chief sources of error in determining the locality of tumours and other local cerebral lesions.—The opinion which is formed as to the position of a tumour may be erroneous for several reasons.

In the *first* place, localising symptoms do not always result from destruction of important cerebral centres. No more striking illustration could be given than the case of destruction of the motor area without paralysis, which is represented in Figs. 1, 2, 3, and 4, pages 11 to 14.

In the *second* place, the new growth may, by altering the relationship of the intracranial contents, and by compressing or stretching the nervous tissues, or by obstructing the blood supply, cause functional disturbances in distant portions of brain tissue (distant centres), and so produce *pseudo-localising* symptoms (see page 15).

In the *third* place, the tumour may in some cases produce *pseudo-localising* symptoms by reflexly stimulating or inhibiting the function of distant parts (centres).

In the *fourth* place, a tumour in one part of the brain may produce symptoms suggestive of a lesion in some other and distant part, by involving the conducting fibres passing to or from that part. Some observers think that tumours or lesions of the angular gyrus produce hemianopsia in this way, viz., by "cutting" (implicating) the radiating fibres of Gratiolet as they pass backwards to the half-vision centre in the occipital lobe. Possibly lesions of the island of Reil produce some forms of aphasia in the same manner, viz., by interrupting the fibres which connect the sensory speech centres (situated in the angular gyrus and first temporo-sphenoidal convolution) with the motor speech centre (situated in the posterior third of the lower frontal convolution—see Fig. 39, page 116).

Vice versâ, lesions (tumours) in the centrum ovale may gradually make their way towards the cortex; and, by causing discharge of cortical centres, may lead the observer to suppose that the tumour is located in the cortex itself. Now, in cases of this description, although the cortex is directly involved by the lesion from below, no diseased appearance may be detectable from the surface, a fact of practical importance; for, when the symptoms clearly show that the function of a given portion of the cortex is deranged, the operator should not rest content with a mere surface view, but should explore the condition of the subcortical tissue (by means of an incision) in those cases in which the surface of the brain appears to be healthy. In Dr. Hughes Bennett's case (the first in which, so far as I know, a cerebral tumour was removed by operation), the superficial appearances of the brain were quite normal; and it was only when an incision was made through the cortex that the tumour was discovered.

At post-mortem examinations it is often difficult to determine the exact point from which the tumour originated, or the part (centre) which was originally invaded. This is one of the practical difficulties which we meet with in trying to determine the function of individual parts of the *human* brain.

In the *fifth* place, intracranial tumours are not infrequently multiple, and in such cases exact localisation may be very difficult, or impossible.

The chief localising symptoms, which have already been considered in detail, are—

1. Symptoms due to deranged function of the intracranial motor nerve apparatus — centres, conductors, and (intracranial) peripheral nerves—such as paralyses, spasms, convulsions, tremors, inco-ordination, motor speech alterations.

These symptoms are of the highest localising value, for our knowledge of the position of the motor centres and the course of the motor-conducting tracts is now far advanced, and the symptoms which result from their derangement are distinct and easily observed. The more limited the paralysis or spasm, the greater of course its localising value. The comparative value of spasms and convulsions as localising symptoms has already been considered (see page 84).

2. Symptoms due to deranged function of the intracranial sensory nerve apparatus—centres, conductors, and (intracranial) peripheral nerves—such as anæsthesia and hyperæsthesia of the limbs, anæsthesia and hyperæsthesia in the area of distribution of the fifth nerve, hemianopsia, word-deafness, word-blindness, &c.

Sensory derangements are, speaking generally, of less localising value than the corresponding motor derangements, for the following reasons:—

(*a.*) They do not, as a rule, so prominently attract the attention either of the patient or the physician. Headache, neuralgia, and hyperæsthetic symptoms generally are, of course, exceptions.

(*b.*) Their exact extent and distribution is, in many instances, more difficult to determine.

(*c.*) They are usually more transitory. Their centres are more widely spread out, and are therefore with more difficulty destroyed. In the cases of the special senses (touch perhaps excepted) the centres are bilateral; substitution is therefore more readily established by the corresponding centre in the opposite hemisphere of the brain carrying on the function of that which is destroyed.

(*d.*) In the case of some of the sense organs, at all events, the sources of peripheral lesion are more numerous. Dimness of vision and deafness are, for instance, more easily produced by peripheral lesions in the eye or ear (end-organs of sight and hearing), than paralysis is by a peripheral lesion of the muscles (motor end-organs). Again, there is

often more difficulty in deciding whether a lesion is peripheral in the case of a sensory than in the case of a motor mechanism. By observing the condition of nutrition, the state of the electrical reactions, and the condition of the reflexes, we can in most cases readily determine whether a paralysis is peripheral or not. It must, of course, be remembered that central lesions involving the nerve nucleus of origin

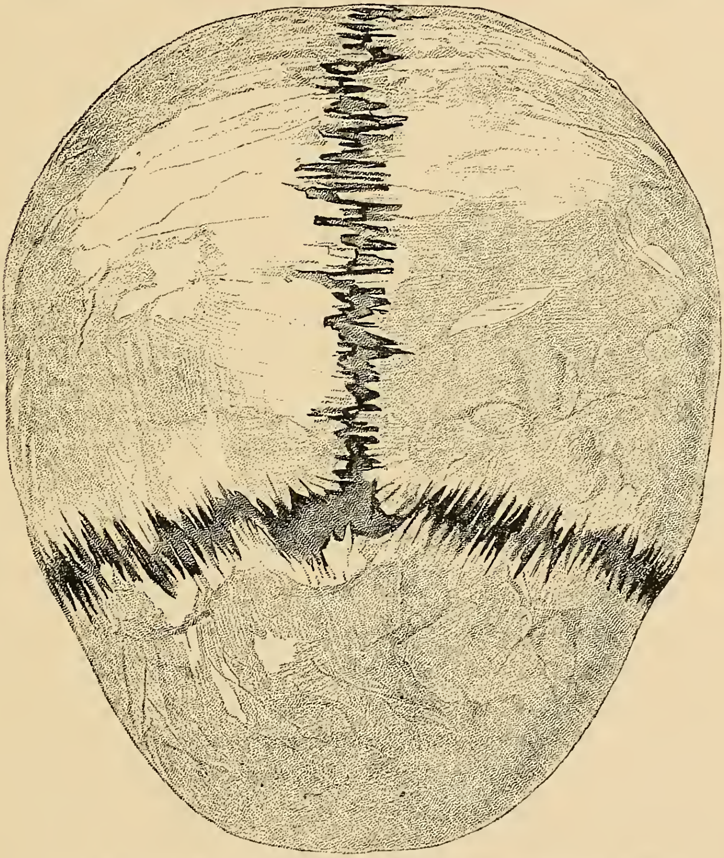

FIG. 45.—The skull-cap seen from above in a case of extreme distention of the ventricles, the result of a scrofulous tumour of the cerebellum. (Copied from a photograph.)

The dark shading between the bones of the skull shows the degree of separation of the sutures.

of a peripheral nerve, produce exactly the same form of paralysis as a lesion of the nerve trunk itself, but the differential diagnosis in such cases is usually not difficult.

3. *The exact nature of the mental symptoms* may sometimes, though it may be confessed rarely, give a clue to the position of the lesion.—(See page 151.)

4. *The presence of certain visceral derangements.*—Tumours of the pons Varolii and medulla oblongata are more likely to be attended with

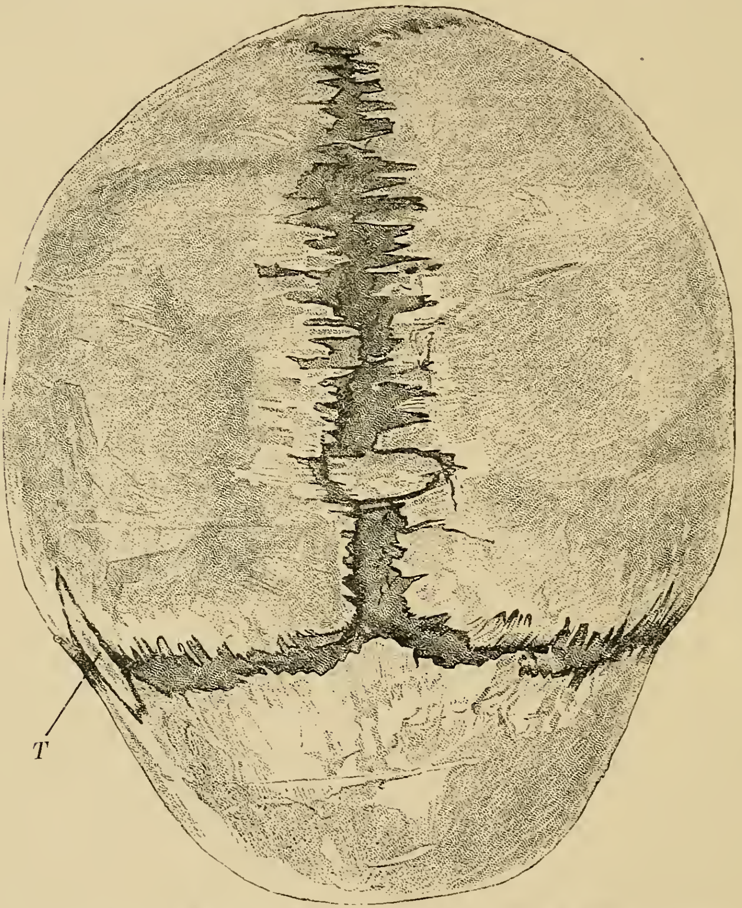

FIG. 46.—The skull-cap seen from above in a case of extreme distention of the ventricles, the result of a hydatid tumour in the cerebellum. (Copied from a photograph.)

The letter *T* points to one of the ossa traquetra, which were largely developed in this case. The dark shading between the bones of the skull shows the extent of separation of the sutures.

visceral derangements (such as polyuria, glycosuria, or even albuminuria, derangements of the pulse and respiration, dysphagia, &c.) than tumours of other parts.

5. *The condition of the cranium and scalp.*—The presence of localised depressions, enlargements, irregularities, or painful spots on the surface of the head, is in some cases of importance. In some cases of cancer and sarcoma the new growth makes its way through the bones of the skull. General enlargement of the head is occasionally seen in children, and is usually due to extensive ventricular dropsy, which is, as we have seen, most frequently associated with subtentorial tumours. A voluminous tumour of one hemisphere may also produce enlargement of the head in a young child. In the adult, after the sutures have become firmly ossified, they do not of course give way; but in young persons, separation of the closed but unossified lines of suture is by no means uncommon.—(See Figs. 45 and 46.)

Let us now consider the symptoms which are produced by, and which are characteristic of, tumours in special parts of the intracranial contents. And in connection with this part of the subject, the localising symptoms only will be specially referred to, but it must of course be understood that in all cases "general" symptoms of different degrees of severity (headache, vomiting, vertigo, double optic neuritis, with or without dimness of vision, &c.) may be present.

Tumours of the Frontal Lobe.

Tumours of the frontal lobe are not uncommon; the nature and severity of the *general* symptoms vary in different cases, and are in no way characteristic; vomiting is perhaps less frequent, and certainly as a rule less severe, than in the case of tumours which are situated further back. Generalised epileptiform convulsions are also perhaps less frequently met with. In many cases the only *positive* symptoms are marked mental impairment, loss of memory, and want of the power of mental concentration. Whether this condition is a constant result of tumours of the frontal lobe remains to be seen. It has been present in all the cases which have come under my own personal observation, and it seems to have been a noticeable result of destruction of the frontal lobes in the lower animals. Mental impairment, loss of memory, and want of the power of concentration are of course frequently produced by lesions in other parts of the brain, more especially "diffused"

lesions. All that we can say is that these symptoms are apparently more marked and more frequently produced by a localised lesion (tumour) which involves the frontal lobes, than by a *localised* tumour which involves other parts of the brain tissue.

In Figs. 47 and 48, the position of two tumours of the frontal lobe, in which mental symptoms were very prominent, is represented.

In one of my cases the head seemed to fall towards the opposite side, apparently as the result of loss of power in the muscles of the neck. This, too, is in accord with Ferrier's observations. He believes "that this portion of the brain is specially concerned in the

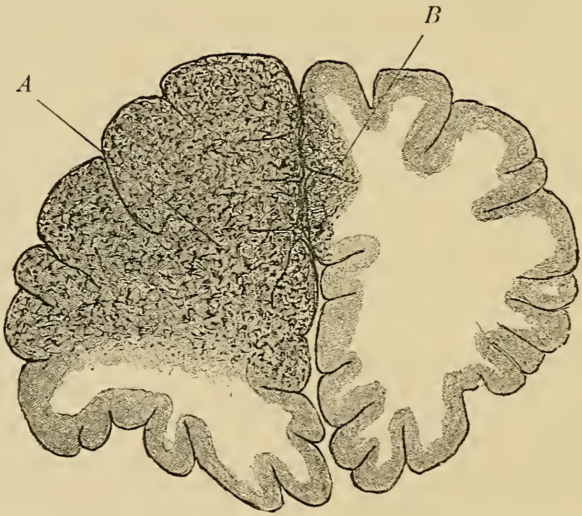

FIG. 47.—Transverse vertical section through the frontal lobes in the case of Miss A., showing the position of the lesion. Drawn from memory. *A* points to the lesion in the left frontal lobe, and *B* to a portion of the right frontal lobe affected by the direct extension of the tumour.

movements of the head and eyes." He noticed that after destruction of the prefrontal lobes a mental deterioration which is essentially a defect of the faculty of attention is observed. He states, "Besides the physiological symptoms, such as occur, and the descending degenerations of the motor tracts associated with lesions of the frontal lobes, I observed and recorded certain symptoms indicative of mental deterioration which have since been confirmed by other physiologists. In my first series of experiments (carried out without antiseptics), I noted, after removal of the prefrontal regions, a decided alteration in

the animals' character and behaviour, but difficult to describe precisely. After the operation, though they might seem to one who had not compared their present with their past, fairly up to the average of monkey intelligence, they had changed considerably. Instead of, as before, being actively interested in their surroundings, and curiously prying into all that came within the field of their observation, they remained apathetic or dull, or dozed off to sleep, responding only to the sensations or impressions of the moment, or varying their listless-

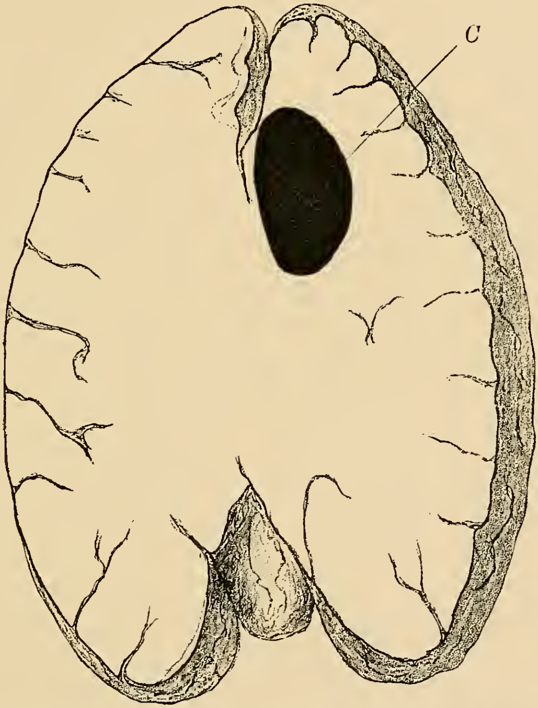

FIG. 48.—Longitudinal section through the brain in the case of D. M., showing the position of the lesion.
The letter *C* points to the tumour.

ness with restless and purposeless wanderings to and fro. Whilst not absolutely demented, they had lost to all appearance the faculty of attentive and intelligent observation." [1]

Tumours springing from the under surface of the frontal lobes, which involve (either directly or by the inflammatory complications

[1] *The Functions of the Brain*, Second Edition, p. 401.

to which they give rise) the olfactory bulbs or tracts, or the optic chiasma, may produce loss of the sense of smell or temporal hemianopsia.

Tumours of the left frontal lobe, which encroach upon or involve the posterior third of the inferior frontal convolution, will in right-handed persons probably be attended with motor aphasia, which, for the reasons previously given, is usually incomplete. Some authorities think that the motor writing centre is situated in the posterior part of the *second* left frontal convolution; and if this is so, tumours of the left frontal lobe, which encroach upon or involve this portion of the cortex, should be attended with motor agraphia. —(See Fig. 39, page 116.)

Tumours confined to the frontal lobes do not produce paralysis (except perhaps impairment of some of the movements of the head and neck, and of the eyeballs), and are not attended with any special sensory disturbances.

Should the tumour extend backwards, so as to involve the fibres passing from the motor centres to the internal capsule, or the motor portions of the internal capsule itself, intermittent tremor, such as is caused by pressure on the motor-conducting fibres (see page 72), or actual paralysis of the muscles on the opposite side of the body, may of course be produced. In the *Edinburgh Medical Journal* for January 1887, page 616, I have reported a case, in which a cyst connected with a glioma of the left frontal lobe was in contact with the motor strands of the internal capsule, and produced "pressure tremor" and intermittent paralysis in the right arm and leg. In two cases of tumour of the frontal lobe, I have observed sudden temporary loss of power in the arm and leg on the opposite side.

Tumours of the Motor Area (Rolandic Region).

Tumours in this situation are as a rule more easily localised than tumours in any other part of the cerebral hemispheres, for the special symptoms which they produce—localised paralyses and spasms—are often most striking and characteristic.

It is, however, important to note that even large tumours which produce extensive destruction of the motor centres may be entirely latent (so far as localising symptoms are concerned), *provided that*

they are of very slow growth.—(See Chapter I., Figs. 1, 2, 3, and 4.) This is more especially the case when the tumour involves the motor centres for the face and trunk.

Tumours involving limited portions of the motor cortex will give rise to localised and limited paralysis or spasm (monoplegia or monospasm), in accordance with the function of the special portion of grey matter (centre) which they irritate or destroy.

General (bilateral) epileptiform convulsions are often caused by tumours in the motor area; but the spasm almost always begins in a definite and localised manner, and without loss of consciousness; in short, the epileptic fit is preceded by a localised epileptiform convulsion of the Jacksonian type (see the case described on page 79.)

When the tumour involves the left motor region, aphasic symptoms are often present.

A progressively advancing paralysis, involving first one part and then another, is very characteristic of a tumour involving the motor cortex or subcortical tissue in this region.

Localised (cerebral) paralysis, without spasm, is more probably due to pressure on, or destruction of, the conducting fibres, than to involvement of the cortical grey matter. It may, however, be due to slow destruction (without initial irritation) of a motor centre.

Localised paralyses of this description are readily enough distinguished from the localised paralyses due to peripheral or nuclear lesions, by the fact that there is no marked atrophy in the paralysed muscles, no "reaction of degeneration," and that, in most cases at all events, the deep reflexes are exaggerated rather than diminished or abolished.

Whether destruction of the motor region of the cortex is attended with any impairment of tactile sensibility, and with loss of the muscular sense in the paralysed parts, is a question which requires further investigation. Ferrier is strongly of opinion that in such cases there is absolutely no anæsthesia; but Charlton Bastian thinks differently, and I have certainly seen some tactile loss in more than one case. It may, however, be confidently stated that lesions (tumours), which involve and are limited to the motor area, do not, so far as we at present know, give rise to marked anæsthesia.

In those cases in which profound anæsthesia (hemi-anæsthesia) is

present, the lesion has in all probability extended from the cortex into the white matter, and invaded the sensory fibres of the internal capsule.

In their later stages, tumours of the motor area may of course extend to and involve surrounding parts. Aphasic symptoms—both motor and sensory—homonymous hemianopsia, and hemi-anæsthesia may be produced in this manner.

With reference to the functions of individual parts of the motor area, see Ferrier's *Functions of the Brain*, and Figs. 16, 17, and 18, pages 72-74; also Horsley and Schäfer's Paper in the *Philosophical Transactions*, 1888.

Tumours of the Post-Parietal Region and Occipital Lobe.

It is now universally acknowledged that the function of those portions of the cerebral hemisphere which are situated behind the Rolandic area (motor region) is sensory; but different observers are not absolutely agreed as to the exact function of the different parts of this extensive region.

The researches of Ferrier seem to show that the angular gyrus and occipital lobe are visual centres, and it would appear that discharging lesions in this region may produce flashes of light or other subjective visual sensations, while destroying lesions may produce hemianopsia. Ferrier's results with regard to lesions of the angular gyrus are, as has been previously stated, disputed by Schäfer (see page 97).

The exact character of the hemianopsia would appear to differ with the position of the lesion. Tumours involving the back parts of the occipital lobe, or the radiating visual fibres as they pass backwards to this centre seem to give rise to the form of homonymous hemianopsia shown in Fig. 23; while lesions (tumours) nearer the angular gyrus (perhaps) produce a form of homonymous hemianopsia in which the peripheral parts of the seeing halves of each field are constricted, and which is represented in Figs. 24 and 25.

Ferrier found that, in the monkey, destruction of the angular gyrus produced crossed amblyopia (blindness of the opposite eye), which was, however, of quite temporary duration. Whether the same condition results from lesions of the angular gyrus in man is not yet determined; but in some hysterical cases (in which perhaps the functional

derangement involves this portion of the cortex) there is, according to Charcot, crossed amblyopia with some peripheral contraction of the visual field in the eye on the side of the lesion (*i.e.*, on the side opposite to the hemi-anæsthesia). Be that as it may, lateral or homonymous hemianopsia seems to result from "destroying" lesions in this situation, and is therefore of some localising value. The importance of a careful examination of the fields of vision by means of the perimeter, in all cases in which there is reason to suspect a tumour of the angular gyrus or occipital lobe, is obvious, and has already been insisted upon.

Since, however, homonymous hemianopsia may also result from a lesion of the optic tract or visual fibres in the internal capsule, it is by no means distinctive of a lesion of the occipital lobe.

If the hemianopsia is preceded by discharges (flashes of light) in the parts of the retina, which subsequently become paralysed, (*i.e.*, blind), as in the case referred to on page 101, the condition is strictly analogous to the ordinary (motor) form of Jacksonian epilepsy, and a (discharging) lesion of the visual centre may reasonably be thought to be present.

Tumours involving the left angular gyrus may in right-handed persons be expected to produce more or less "word-blindness."

In addition to these visual defects, tumours of the occipital lobe and post-parietal region often produce impairment of the sense of touch on the opposite side of the body, for they frequently involve the conducting fibres, which convey tactile impressions, as they radiate from the posterior part of the internal capsule to the tactile centre in the cortex. The exact position of the tactile centre is still a disputed point, but the observations of Ferrier and Schäfer seem to show that it is situated in the hippocampal region and gyrus fornicatus (see page 89). Whether the tactile sense is in any way represented in the convolutions of the Rolandic (motor) area is also, as we have seen, a matter of debate.

Tumours of the post-parietal region may of course extend forwards and involve the motor centres, or downwards and inwards, and involve the motor-conducting fibres as they pass from the motor centres to the motor region of the internal capsule. Spasms and paralysis may therefore be present in the later stages of such cases.

General epileptiform convulsions, preceded perhaps by a sensory

(visual) aura, and not resulting from a localised motor discharge (in other words, not preceded by ordinary Jacksonian epilepsy), are perhaps more common in cases of tumour of the post-parietal and occipital regions than in tumours of the other parts previously described (frontal lobe and motor area).

Dr. Hughlings Jackson seems to think that lesions of the occipital lobes, and especially of the right occipital lobe, are attended with more profound mental impairment than lesions of the motor area, but this opinion is doubted by some other authorities. My own experience would indeed lead me to suppose that the mental deterioration is greater when the tumour involves the frontal, than when it involves the occipital lobes.

Tumours of the Temporo-Sphenoidal Lobe.

In these cases there are often no special symptoms. The centres for hearing, taste, and smell are situated in the temporo-sphenoidal lobe, but, as has been previously stated, marked loss of hearing, taste, and smell does not seem to result from destruction of the centre on one side. It is probable, however, that complete destruction of the hearing centre (and perhaps also of the smell and taste centres) on one side *is* attended with some degree of impairment.

Lesions of the upper temporo-sphenoidal convolution, on the left side (auditory " word centre "), in some cases at all events, seem to produce " word-blindness."

Epileptiform convulsions, preceded by a noise in the ear, or by a peculiar taste or smell, are suggestive of a discharging lesion of the corresponding centre in the temporo-sphenoidal lobe.

Tumours which destroy the hippocampal region may be expected to produce hemi-anæsthesia, and (according to Ferrier) loss of the muscular sense on the opposite side of the body. Tumours of the temporo-sphenoidal lobe, which do not directly involve the hippocampal lobule (the tactile centre), may interrupt or destroy the tactile conducting fibres as they pass to that centre.

In some cases hysterical symptoms are prominent, but whether this is merely accidental, or the result of the special position of the lesion (in the temporo-sphenoidal lobe), I am unable to say.

Large tumours of the temporo-sphenoidal lobe may extend to and

invade other and adjacent centres or tracts, and in their later stages may be attended with spasms, paralyses, visual defects, motor speech derangements, &c., from invasion of the motor area or post-parietal regions.

Tumours of the Centrum Ovale.

The character of the localising symptoms in these cases depends upon the exact position of the tumour and its relationship to the special fibres and tracts of which the centrum ovale is composed. Tumours of the front part of the centrum ovale chiefly involve the fibres passing downwards from the frontal lobe; tumours of the mid-region those from the motor centres; and tumours of the posterior part the fibres passing upwards to the sensory centres in the occipital and temporo-sphenoidal lobes.

The nearer the lesion is to the basal ganglia and internal capsule the greater the risk of all three sets of conducting fibres being involved.

Lesions in the centrum ovale will also, of course, intercept the fibres which connect the two hemispheres, and different parts of the same hemisphere with one another. Our knowledge of the course and functions of these connecting (commissural) fibres is, as yet, so entirely indefinite, that it is impossible to make any statement as to the special symptoms which result from their destruction. It may, however, be stated that tumours of the left hemisphere, which destroy the fibres connecting the sensory with the motor speech centres (the exact course of which is not yet definitely determined), may be expected to produce aphasic symptoms.

Tumours of the centrum ovale, which involve the cortical grey matter, may act as sources of irritation and discharge, and may produce spasms, convulsions, or sensory phenomena, but in most of the cases of this kind, which have come under my own notice, irritative symptoms have not been prominent.

The absence of irritative symptoms in the cases to which I refer may have been due to the fact that the tumours were mostly gliomata, which are, I think, less apt to excite discharge than some other forms of new growth; and to the circumstance that many of the conducting fibres, passing from the cortical centres in contact with the new growth, were destroyed before the centres themselves were irritated.

Tumours of the Corpus Callosum.

Tumours in this situation are not very common. They are usually gliomata or sarcomata, which steadily progress until the new growth may make its way into the centrum ovale of each hemisphere. The symptom which is most characteristic of a tumour in this situation is a gradually developing paralysis (more or less complete hemiplegia), first on one side and then on the other. Should the tumour invade the posterior part of the centrum ovale rather than the anterior, there might be a gradually advancing and bilateral hemianæsthesia instead of motor paralysis.

There is, in addition, in some cases, marked mental impairment, and some affection of speech. Dr. Bristowe, who has written the most complete account of tumours of the corpus callosum with which I am acquainted, sums up their chief characters as follows :—

"1*st*. Their ingravescent character—a character which they possess in common with other cases of cerebral tumour; 2*nd*. The gradual coming on of hemiplegia, for the most part resembling in its distribution the paralytic symptoms usually attending hæmorrhage into one of the hemispheres, or softening due to embolism ; 3*rd*. The association with the paralysis of one side of vague hemiplegic symptoms of the other; 4*th*. The supervention of stupidity, associated for the most part with extreme drowsiness, a puzzled inquiring look when awake, a difficulty of getting food down the throat, and cessation of speech. I say a difficulty of getting food out of the mouth, rather than paralysis of the mouth and throat, and I say a cessation of speech rather than aphasia or loss of articulating power, because it seems to me, in watching the cases, that these phenomena were due mainly to stupidity and irresistible tendency to sleep, and not definitely to paralysis or affection of the centres for speech; 5*th*. The absence of implication of the oculo-motor nerves, and of direct implication of other cerebral nerves; and lastly, death from coma." And again—"Indeed I take it that the symptoms the patients presented were due chiefly, if not altogether, to the extension of the disease into the hemispheres and to the diffused pressure on important parts caused by the great collective bulk of the tumour, as evidenced by the extensive flattening of the surface of the hemispheres, and the

singular absence of both subarachnoid and ventricular fluid. Assuming this explanation to be correct, the progress of a case of the disease I have been describing should be as follows:—*first*, the occurrence of headache and other somewhat vague symptoms of progressive cerebral disease; *second*, the gradual onset of more or less well-marked hemiplegia; *third*, the appearance, in a greater or less degree, of similar symptoms on the opposite side of the body; *fourth*, the coming on of dementia, with drowsiness, loss of speech, difficulty in swallowing, and want of control over the rectum and bladder."[1]

Tumours of the Base.

The new growths which most frequently occur at the base of the brain are—syphilitic gummata; tumours (simple enlargement or various forms of new growths) of the pituitary body; aneurisms springing from the arteries forming the circle of Willis; and cancerous and sarcomatous tumours growing from the bones at the base of the skull, or from the dura mater.

The character of the symptoms depends upon the exact position of the new growth; but the most important localising symptoms are due to pressure on, or involvement of, one or more of the cranial nerves, on one or both sides. When several nerves are implicated, the exact position of the tumour can be more accurately determined than when only one is involved, for implication of several nerves means a lesion at the point where they come in contact or juxtaposition. Paralysis, for example, of the parts supplied by the third, fourth, and sixth nerves on one side, with unilateral optic neuritis or optic atrophy, means a lesion either in the back of the orbit or involvement of the nerves as they pass into the orbital fissure and optic foramen; while paralysis of the external rectus may be produced by many different lesions, involving the sixth nerve in its long and exposed course.

Tumours of the anterior region of the base.—Tumours of the pituitary body, when of large size, such as that represented in Figs. 49 and 50, may be attended with general symptoms (headache, vomiting, optic neuritis, gradual and progressive dementia); but in some cases the general symptoms are little marked. Aneurisms in this situation are often "latent" until the final rupture occurs; for the

[1] *Brain*, vol. vii., pp. 318 and 319.

majority of aneurisms at the base of the brain are of small size. It is altogether exceptional for them to attain to the extraordinary dimensions shown in Figs 51, 52, 53, 54, and 55.

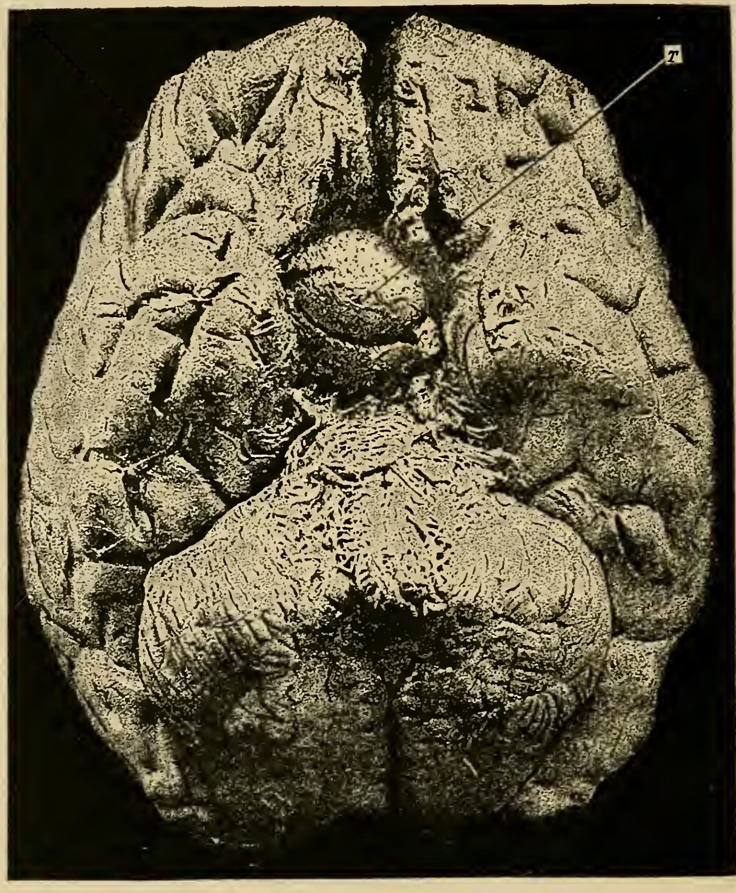

FIG. 49.—Autotype reproduction of a photograph of the base of the brain, showing a tumour of the pituitary body. (Considerably less than the actual size.)

A brief record of the case will be found in Dr. Berry's *Subjective Symptoms in Eye Disease*, page 111. There was very marked temporal hemianopsia, the temporal halves of each field being absolutely blind.

The most important localising symptom of tumours in the position of the pituitary body is probably temporal hemianopsia, the result of pressure on the central part of the optic chiasma. It is important to remember that, in cases of this description, optic neuritis may be absent, and that after the hemianopsia has lasted for a longer

or a shorter time, optic atrophy and total blindness (not preceded by papillitis) may be developed.

In some cases the defect of vision varies in extent from time to time, being better one day and worse the next. Such alterations are suggestive of fluid pressure, such as would be caused by an aneurism or cyst, the tension of which was variable.

The same alterations in the extent of the visual defect have, however, been noticed as the result of syphilitic lesions (gummata) in this situation.

Tumours in the region of the pituitary body may involve one or both olfactory tracts, and produce loss of smell in one or both nostrils.

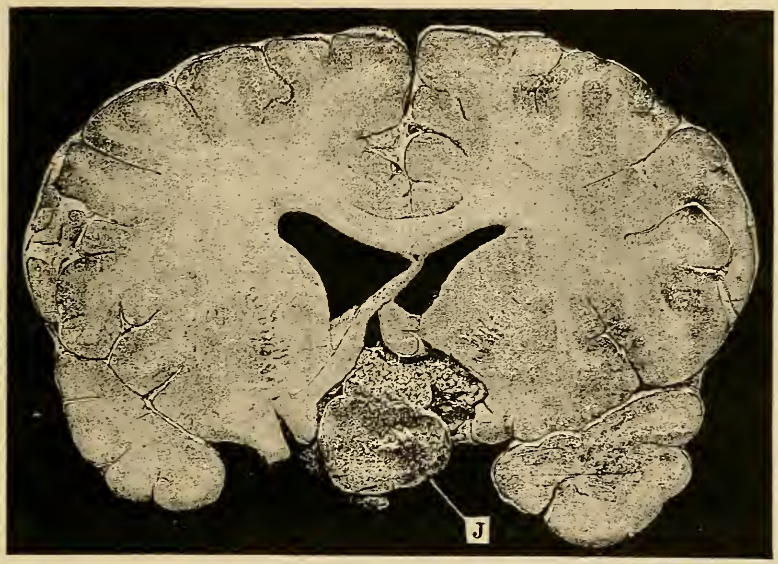

FIG. 50.—Transverse vertical section through the brain in the case represented in Fig. 49, showing the relationship of the tumour (*J*) to the adjacent parts. (Facsimile representation of a section of the brain prepared by the author's "ready method.")

The tumour is growing upwards towards the ventricles; and has produced considerable distortion of the parts with which it is in contact.

The third, fourth, and sixth nerves on one or both sides may also be implicated.

Tumours of large size (such as those represented in Figs. 49 to 55) may press upon the crura cerebri, and make their way upwards towards the ventricles. Dementia and hebetude may in such cases be very great. Paralysis, usually incomplete, in the face or limbs on one

or both sides may also result from the pressure of the tumour on the motor tracts.

Tumours of the pituitary body are in many instances attended with

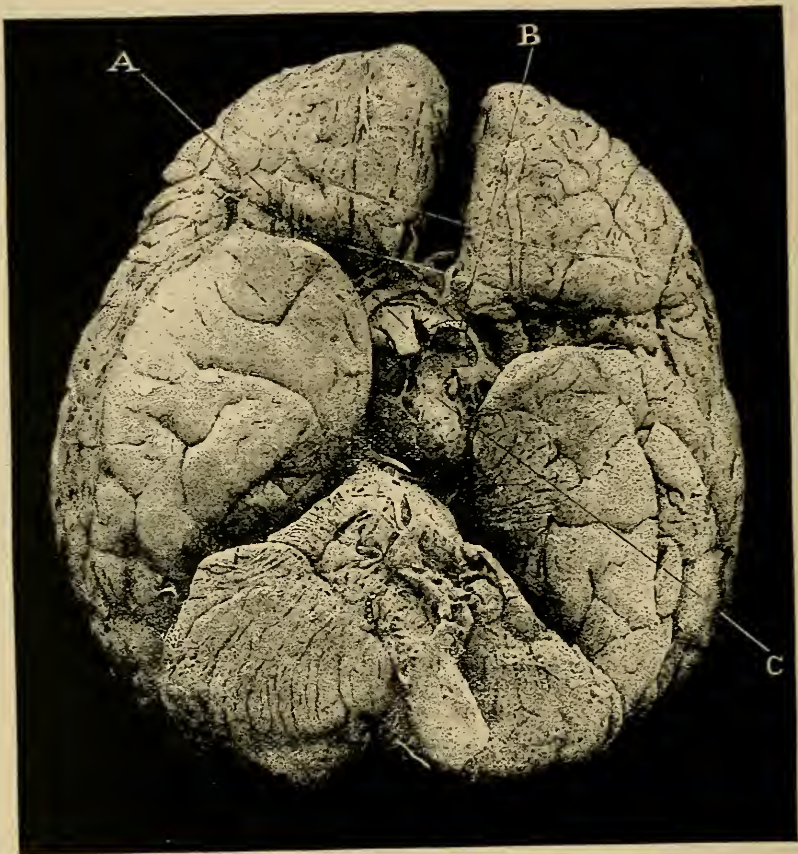

FIG. 51.—Autotype reproduction of a photograph of the base of the brain in the case of C. F., showing the aneurism (*C*) projecting downwards in the interpeduncular space. (Considerably less than the actual size of the preparation.)

The right hemisphere of the brain and the left lateral lobe of the cerebellum are smaller than the corresponding parts on the opposite side. Whether this difference was present during life it is difficult to decide. In consequence of the division of the corpus callosum and the long preservation in spirit, the relative position and relationship of the parts were somewhat altered. Before this photograph was taken the frontal lobes were fixed in position by means of a thin piece of string. The aneurism does not project downwards in the hardened brain (photograph) so far as it appears to have done in the recent state. The letter *A* points to the right, and the letter *B* to the left optic nerve, which are twisted backwards. Immediately above the part of the aneurism to which the letter *C* points, the left internal carotid artery, with a clot projecting from its cavity, is situated.

an excessive development of the subcutaneous fat, and in some cases with the presence of sugar in the urine, or with simple polyuria (diabetes insipidus).

Whether these symptoms are due to the fact that the pituitary body itself is diseased, or whether, as seems more likely, to the secondary results which tumours in this situation produce in the surrounding cerebral tissue, has not yet been decided.

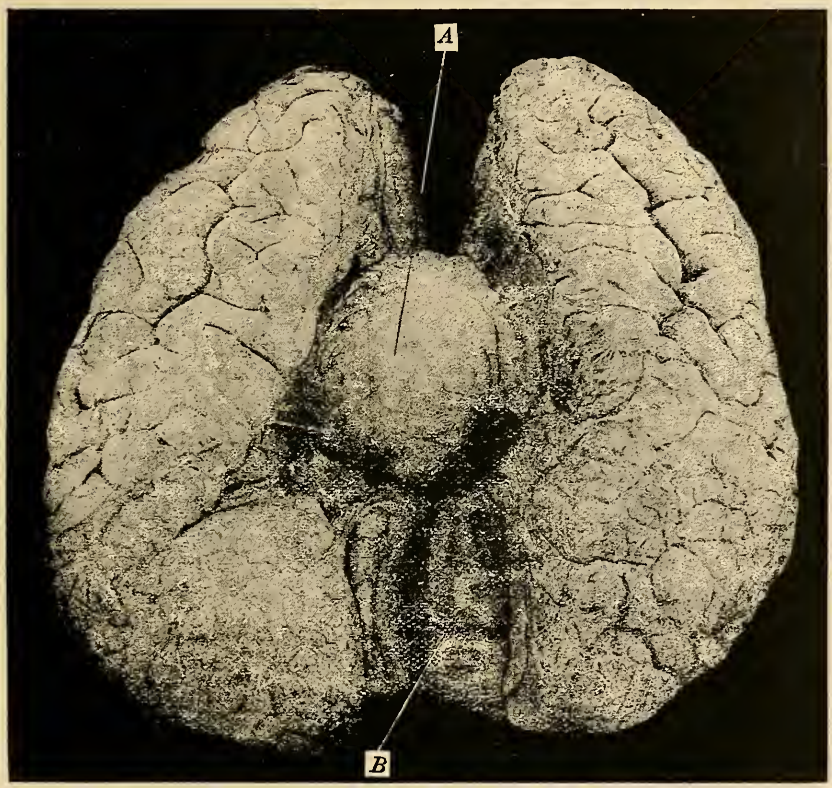

Fig. 52.—Autotype reproduction of a photograph of the brain in the case of C. F., showing the aneurism projecting upwards into the third and lateral ventricles. (Considerably less than the actual size of the preparation.) The corpus callosum has been divided and the hemisphere separated so as to fully expose the tumour; the upper surface of the cerebellum has also been partly divided in an antero-posterior direction. The letter *A* points to the aneurism; *B*, to the right side of the upper surface of the cerebellum.

Possibly, as Rosenthal has suggested, the diabetes, which is sometimes present, may be the result of secondary changes produced in

the grey matter of the floor of the fourth ventricle. He supposes that the pressure of the tumour first produces irritation of the grey matter lining the third ventricle, and that this irritation travels along the grey matter which connects the third and the fourth ventricle, and produces secondary changes in the latter. "According," he says,

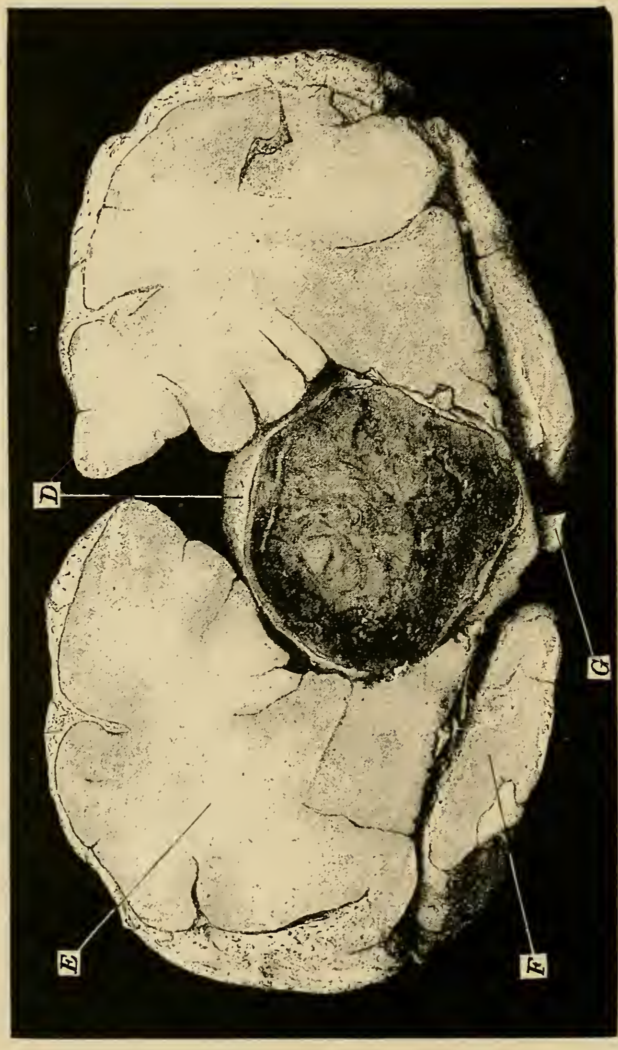

FIG. 53.—Autotype reproduction of a photograph of a transverse vertical section through the brain in the case of C. F., showing the maximum dimensions of the aneurism. (Slightly less than the actual preparation.)

The photograph does not accurately represent the relationship of the parts during life, for the corpus callosum, which was during life stretched across the upper surface of the tumour, has been cut through, and its two divided ends are widely separated in the figure. The letter *D* points to the upper surface of the aneurism; *E*, to the white matter of the left hemisphere; *F*, to the left temporo-sphenoidal lobe; *G*, to the right optic nerve.

"to the experiments of Claude Bernard, lesions of the floor of the fourth ventricle will cause the appearance of sugar in the

urine (from lesion of the centre of the vascular nerves of the kidney according to Schiff). Tumours of this region may also produce glycosuria. If we consider, in addition, that the tuber cinereum is found in the immediate neighbourhood of, and in advance of the pituitary gland, and that the infundibulum merely represents a pro-

Fig. 54.—Autotype reproduction of a transverse vertical section through the frontal lobes in the case of C. F., showing a cavity (*C*) into which the aneurism projected. (Slightly less than the actual size.)

The case represented in Figs. 51, 52, 53, and 54 occurred in the practice of Dr. Carlyle Johnstone, and that represented in Fig. 55 in the practice of Dr. Clouston.

I am indebted to these gentlemen, not only for the specimens, but also for kindly permitting me to record the cases. (See *Edinburgh Medical Journal*, April 1887, p. 911.)

longation of the grey substance of the third ventricle, and by continuity of the fourth ventricle into the medulla oblongata, we may admit that tumours of the pituitary region, by an increase of pressure, may give rise to a paralysis of the medullary centres of hepatic innervation, and to consequent hyperæmia of the liver, resulting in diabetes ?"[1]

[1] *Diseases of the Nervous System*, American translation, p. 120.

Probably some of the symptoms which result from tumours in the region of the pituitary body are due to the pressure of the tumour on the great vessels at the base of the brain (branches of the circle of Willis), and to the alterations in the nutrition of the cerebral tissues which may result therefrom.

Syphilitic gummata are very common in the neighbourhood of the interpeduncular space. The general symptoms to which they give rise are usually very marked. The special localising symptoms are due either to involvement of the nerves (such as the third), to asso-

FIG. 55.—Autotype reproduction of a photograph of an immense intracranial aneurism of the right internal carotid artery. (Actual size after long preservation in spirit.)

ciated arterial disease (with resulting softening of the cerebral tissue, sometimes at a considerable distance from the seat of the primary lesion), or to pressure and involvement of adjacent parts, such as the crus cerebri. Lesions in this situation may therefore produce that form of alternate hemiplegia, in which the muscles supplied by the third nerve are paralysed on the same, and those of face, arm, and leg on the opposite side of the body, in two ways, viz., (1.) by involving

the trunk of the third nerve and the motor tract for the face, arm, and leg, in the crus cerebri; or (2.) by involving the trunk of the third nerve and the middle cerebral artery (and producing softening of the motor tract in the higher parts of the cerebral hemisphere).

The mode of onset of the paralysis may be different in the two cases. In the latter case the paralysis of the eye muscles on the same side, and of the face and the limbs on the opposite side, are not, as a rule, developed so simultaneously as in the former.

The sudden occurrence of this form of alternate hemiplegia, therefore, indicates a lesion of the crus cerebri.

Paralysis of the muscles supplied by the third nerve, followed, after a longer or a shorter interval, by hemiplegia on the opposite side, *may* be due to a gumma at the base involving the third nerve and middle cerebral artery. A paralysis of the muscles supplied by the third nerve, closely followed by a slowly developing paralysis of the face, arm, and leg on the opposite side, is probably due to a lesion at the base, involving the trunk of the third nerve, and then pressing upon and invading the crus cerebri.

Syphilitic gummata, or other tumours in the region of the interpeduncular space, may invade one optic tract, but in many cases the chiasma and one or both optic tracts, or the chiasma and one or both optic nerves, are involved.

Tumours of the middle region of the base.—The most characteristic symptoms suggestive of a tumour in the middle fossa are those which are due to implication of the trunk of the fifth nerve or the Gasserian ganglion, viz., neuralgic pains in *all* the areas of distribution of the nerve, or anæsthesia of the same parts; spasmodic contraction or rigidity, but much more frequently paralysis, of the masseter and temporal muscles; and, in some cases, trophic alterations in the eyeball—inflammation and suppuration and ulceration of the cornea.

The trunk of the seventh and eighth nerves (auditory and labyrinthine portions) may also be involved, with resulting facial paralysis, deafness, or giddiness.

The third, fourth, and sixth nerves, the optic tracts, crus cerebri, and the other parts, which are apt to be involved by tumours in the region of the pituitary body or interpeduncular space, may also, of course, be implicated.

Tumours of the posterior region of the base.—Tumours of the posterior fossa, which are situated laterally, are apt to involve the trunk of the fifth nerve at its origin from the pons, and so to produce neuralgia or anæsthesia in its area of distribution, spasms or paralysis of the masseter and temporal muscles, and rarely (much less frequently than when the Gasserian ganglion is involved) trophic alterations in the eyeball. Tumours in this situation are also apt to involve the seventh (facial), eighth (auditory and labyrinthine), ninth, (glosso-pharyngeal), tenth (pneumo-gastric), eleventh (intracranial portion of the spinal accessory), and twelfth (hypoglossal) nerves, and therefore to be attended with symptoms suggestive of a lesion of the pons Varolii or medulla oblongata.

Tumours of the posterior fossa, which are centrally situated, may involve one or both sixth nerves, and, by pressing upon the anterior pyramids of the medulla oblongata, are more apt to produce motor symptoms in the limbs than those tumours of the posterior fossa, which are more laterally situated.

The differential diagnosis of tumours within the pons Varolii or medulla oblongata, and of tumours at the base, pressing upon the pons or medulla from without, is not always possible. In the latter case the cranial nerves are more frequently involved before the paralysis or anæsthesia in the arms and legs is established. The same condition of matters may, however, result from a tumour within the pons. Implication of several nerve trunks (in the posterior fossa) without limb paralysis is, however, strongly in favour of a lesion at the base, outside (rather than inside) the pons Varolii or medulla oblongata. A small tumour outside the pons might involve the trunk of the hypoglossal or ninth nerve on the same side, and the pyramidal tract, proceeding to the opposite arm and leg. It will be observed that in this form of alternate hemiplegia the face may not be affected, for the lesion is situated below the point of exit of the facial (seventh) nerve.

The manner, too (the sequence), in which the different nerves are involved is of importance, for nerves, which on the surface or at the base of the brain are situated closely together, often diverge within the pons or medulla in making their way to or from their respective centres (nerve nuclei) of deep origin. At the base of the brain

(outside the pons) the seventh and eighth nerves are, for example, so closely placed together that it is almost impossible for a tumour to press upon and involve one without also implicating the other; but within the pons, a small lesion may easily do so.

Tumours of the Basal Ganglia.

New growths which invade the basal ganglia are rarely limited to these structures, but usually involve the internal capsule or other adjacent parts. Even in those cases in which (as in that represented

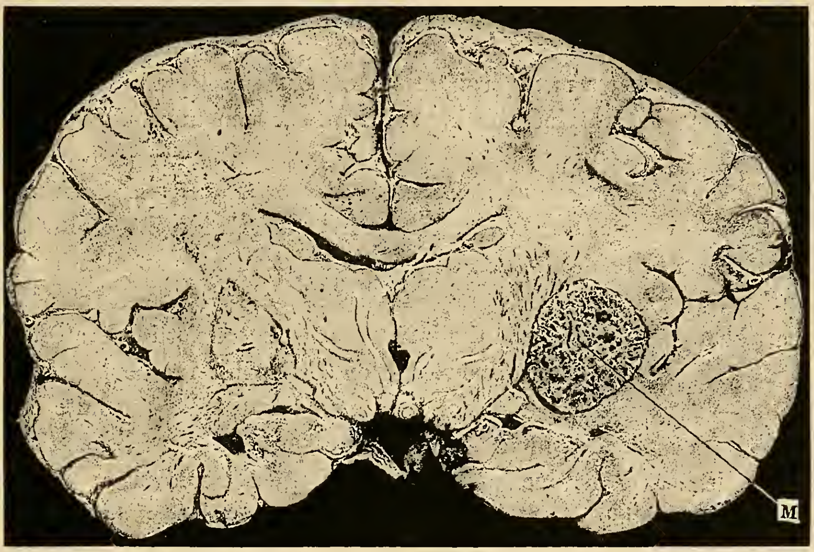

Fig. 56.—Autotype representation of a transverse vertical section through the brain in a case of secondary cancer, showing a cancerous nodule (*M*) in the position of the lenticular nucleus, which was in fact completely destroyed by the new growth. (Reduced from a photograph.)

in Fig. 56) a nodule of new growth is limited to one or other of the basal ganglia, other nodules are often present in other parts of the intracranial nerve centres; the clinical picture is consequently complicated, and it is difficult or impossible to determine the symptoms which are due to the lesion of the ganglia themselves.

But, notwithstanding these difficulties, it would appear (more especially from a study of the effects of the hæmorrhagic extravasations and softenings, which are much more frequently limited to

the basal ganglia than new growths) that destruction of these structures is not attended with symptoms of a striking or permanent character.

The localising symptoms which new growths involving the basal ganglia do produce, seem to be for the most part due to pressure on, or involvement of, the motor and sensory fibres of the adjacent internal capsule, and in some cases of the optic tract. They consist of hemiplegia and hemi-anæsthesia on the opposite side of the body, and of homonymous hemianopsia. It is of course theoretically possible, though practically unlikely, that a tumour originating in one or other of the basal ganglia might involve, for a time at least, the bundles of fibres for the face, arm, or leg individually, and might therefore produce a monoplegia or imperfect hemiplegia, which, with the development and increase in the size of the tumour, would become more and more complete.

Tumours of the corpus striatum (nucleus caudatus and lenticular nucleus).—So far as can be ascertained from the observation of disease in man, complete destruction of the nucleus caudatus and lenticular nucleus is unattended by any characteristic symptoms, provided that the adjacent strands of the internal capsule are not implicated.

Ferrier sums up our knowledge on this point in the following words—"As a rule the lesions of disease are more or less indefinite, but the careful investigation of Charcot and others have established that, though lesions of the nucleus caudatus or lenticular nucleus appear to cause hemiplegia, in all respects similar to that caused by lesions of the anterior two-thirds of the internal capsule, or of the cortical centres themselves, yet the hemiplegia depending on lesions limited to the grey matter of the striate nuclei is of a transitory character, and may entirely disappear even while the lesion remains."

I have myself reported a case of multiple scrofulous tumours, in which almost the whole of the nucleus caudatus on the left side was destroyed by a nodule of new growth, and in which there was no paralysis,[1] and another case in which the lenticular nucleus was completely destroyed, and in which paralysis could not be detected during life.[2]

[1] *Edinburgh Medical Jonrual*, February 1879, p. 697.

[2] *Brain*, January 1888, p. 503.

Whether irritative lesions of the corpus striatum produce any characteristic symptoms has yet to be determined. Ferrier finds that "electrical irritation of the intra-ventricular portion of the nucleus of the corpus striatum causes tonic contraction of the whole of the muscles on the opposite side, resulting in a condition of pleurosthotonus, in which the position assumed is that of equilibrium between the flexors and extensors," and, with good reason as it seems to me, he thinks that these spasms are due to irritation of the grey matter of the ganglion itself, rather than to irritation of the adjacent fibres of the internal capsule.

Nothnagel found that irritation (mechanical and chemical) of a localised spot in the head of the corpus striatum (nucleus caudatus) produced an irresistible tendency to run or jump, which movements continued until the animal became exhausted; the portion of grey matter which was irritated in these experiments he has consequently termed the nucleus cursarius.

Destruction of the lenticular nucleus by the same means (injections of chromic acid) was found by Nothnagel to produce marked disturbances, viz., outward deviation of the opposite fore-leg, and inward deviation of the hind leg on the same side, bending of the vertebral column towards the side of the lesion, and some dorsal curvature due to paralysis of the trunk muscles. "When the lenticular nuclei were destroyed on both sides there was no deviation of the limbs nor distortion of the trunk. The animals maintained their normal attitude, but remained quite immoveable and apathetic, like animals deprived of their cerebral hemispheres. They allowed their limbs to be withdrawn or placed in any abnormal position without resistance; but if the tail was pinched, the animal would make one or two leaps forward, and again relapse into its apathetic immobility."[1]

Ferrier seems to doubt the value of these observations, and to think that the results which Nothnagel obtained may have been due to lesions of the adjacent fibres of the internal capsule.[2]

Tumours of the optic thalamus seem in some cases to produce disturbances of vision. Some writers state that lesions limited to the optic thalamus may be attended with homonymous hemianopsia, but

[1] Quoted from Ferrier, *Functions of the Brain*, Second Edition. p. 411.
[2] *Ibid.*, p. 416, sect. 8.

in most cases of this kind the visual defect was in all probability due either to involvement of the external geniculate body, or to pressure upon the optic tract.

In athetosis and post-hemiplegic chorea the lesion is frequently situated in the optic thalamus, and it is conceivable that these symptoms might therefore be produced by a tumour in this situation.

Besides the ordinary form of hemiplegia and hemi-anæsthesia, which lesions (tumours) of the optic thalamus may produce by involving, or pressing upon, the adjacent fibres of the internal capsule, it is possible that a tumour in this situation might produce the form of alternate hemiplegia which is usually due to a lesion of the crus cerebri, in which the muscles supplied by the third nerve are paralysed *on the same side* as the lesion, while the muscles of the face, arm, and leg are involved on the opposite side.

Tumours of the Pons Varolii, Corpora Quadrigemina, and Pineal Gland.

Small tumours situated in the middle of the pons Varolii, and those new growths which do not involve the great conducting (pyramidal) tracts, or the nerves arising from the pons (the nerve roots or nerve nuclei), may, for a time at all events, be unattended by any characteristic symptoms.

General symptoms (headache, vomiting, and optic neuritis) are usually present, but by no means always prominent.

In most cases of tumour of the pons Varolii there are, however, well-marked localising symptoms, the exact character of which varies in different cases, and depends upon the special part of the pons in which the tumour is situated.

These symptoms consist of—(1) paralysis and anæsthesia of the face, limbs, and trunk, the result of involvement of the fibres of the pyramidal tracts as they pass through the pons; and (2) paralysis or anæsthesia due to implication of the cranial nerves which spring from the pons, or of their root-fibres, nerve-nuclei, or connecting fibres (third, fourth, fifth, sixth, and seventh nerves).

The paralysis and anæsthesia may be on one or both sides of the body.

Tumours which implicate the pyramidal tract on one side, in the

upper third of the pons (*i.e.*, above the point at which the fibres of the facial nerve leave the pyramidal tract to decussate and pass to their nerve nucleus on the opposite side), produce ordinary hemiplegia, *i.e.*, paralysis of the face, arm, and leg, on the opposite side (see 6, Fig. 57).

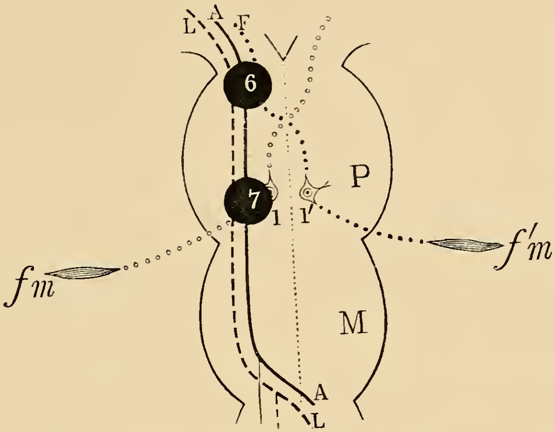

FIG. 57.—Diagram showing the distribution of the paralysis which may result from lesions in the upper and lower parts of the pons Varolii.

The letters *F*, *A*, *L* point to the motor tracts for the left side of the face, arm, and leg, as they enter the right side of the pons Varolii (*P*) in their passage from the right hemisphere of the brain. (Compare with Fig. 14, p. 69.)

A lesion in the upper third of the right half of the pons, in the position of 6, will cause paralysis of the face, arm, and leg on the opposite (left) side—ordinary hemiplegia.

A lesion in the lower third of the right half of the pons, in the position of 7, will cause that form of alternate hemiplegia in which the face is involved on the same (right), and the arm and leg on the opposite (left) side.

If the sensory fibres of the pyramidal tract are at the same time involved, hemi-anæsthesia, involving the face, arm, and leg on the opposite side, is also present.

Tumours in this situation (in the lateral half of the upper third of the pons) may also involve the fibres or nerve nuclei of the third or fourth nerves *on the same side*,—in short may produce the form of alternate hemiplegia which is so suggestive of a lesion of the crus cerebri.

Tumours at the top of the pons Varolii, and tumours involving the corpora quadrigemina, are comparatively rare. In addition to the general symptoms suggestive of an intracranial tumour which are

usually present, the special symptoms which we would expect a lesion in this situation to produce, are paralysis of the ocular muscles supplied by the third and fourth nerves, disorders of co-ordination and inability to maintain the equilibrium, tonic spasmodic contractions of the muscles of the limbs and trunk, and perhaps (when the anterior tubercles of the corpora quadrigemina are involved) some disorders of sight. Ferrier concludes, as the results of his experiments, that "the optic lobes are the centres of correlation between retinal impressions and oculo-motor reactions," and that, "whatever may be their exact position, we are justified in assuming that there are, in the region of the corpora quadrigemina, fibres of connection between the optic tracts and the oculo-motor nuclei, and that the integrity of the connections is a necessary condition of reflex irido-motor and general oculo-motor reaction." Like other physiologists, he finds that "lesions of the corpora quadrigemina in various animals give rise to marked disturbances of equilibrium and irregularity of movement," and he states that "entire removal of the optic lobes renders station and locomotion impossible." Further, he found that electrical irritation of the corpora quadrigemina is followed by well-marked and definite phenomena, and that irritation of the nates, or anterior pair of tubercles, produces different results from irritation of the testes. These phenomena were as follows:—"On irritation," he says, "of the nates, or anterior tubercles, in monkeys, I have observed the following results:—Irritation of the one side causes the opposite pupil to become widely dilated, followed almost immediately, or accompanied by, dilatation of the pupil on the same side. The eyes are widely opened and the eyebrows elevated; the eyeballs are directed upwards and to the opposite side; the head is moved in the same direction as the eyes; the ears are strongly retracted. If the irritation is kept up, the tail is elevated, the legs extended, the arms approximated to the sides, drawn back and flexed at the elbows, the jaws clenched, and the angles of the mouth retracted, until a general opisthotonus ensues. The motor effects are first shown on the opposite side of the body, but ultimately both sides become affected by the unilateral irritation.

Irritation of the testes, or posterior tubercles, causes the same dilatation of the pupils and general motor symptoms, but with these, and as the first effect, the utterance of a short bark or cry on the slightest

contact of the electrodes, and every variety of vocalisation when the stimulation is continued."

"The phenomena," he states, "which result from irritation, mechanical, chemical, or electrical, of the lamellæ of the corpora quadrigemina or optic lobes, appear to be mainly, if not exclusively, of a reflex character, and are such as may result from irritation of sensory centres or tracts. If we assume that in the corpora quadrigemina sensory impressions, retinal and others, are co-ordinated with adaptive motor reactions, such as are involved in equilibriation and locomotion, we should scarcely expect to excite these otherwise than in a tumultuous and ungraduated manner by irritation applied to the centres themselves. . . . So also the dilatation of the pupils, which is so readily induced by irritation of the corpora quadrigemina, may be regarded as a sign of irritation of sensory structures. . . . No direct relation has been satisfactorily established between irritation of the nates and the constringent action of the irido-motor nucleus of the third nerve. This is a fact which favours the views of those who hold that the corpora quadrigemina as such are not the medium of the irido-constrictive reaction induced by stimulation of the retina."

"The phenomena," he states, "occurring on irritation of the nates and testes respectively appear to me to be the result of irritation of these ganglia as such, and not of the subjacent tracts, and to depend upon the structural differences between the anterior and posterior pair of tubercles."

He sums up his opinion as follows :—"From the phenomena occasioned by electrical or other forms of irritation of the corpora quadrigemina or optic lobes, it would appear, therefore, that nothing is indicated with any degree of certainty further than the existence of retinal and other sensory tracts or centres, through stimulation of which general motor reactions are capable of being induced. In so far, however, they support the hypothesis, founded on the effects of destructive lesions, that these ganglia form an essential portion of the mechanism of the co-ordination of retinal and general sensory impressions with the mesencephalic motor apparatus concerned in the complex responsive adjustments of equilibriation and the other adaptive reactions of which animals are capable after removal of the higher encephalic centres."[1]

[1] *The Functions of the Brain*, Second Edition, Chapter V.

When the adjacent motor or sensory tracts are involved, there may of course be paralysis and anæsthesia of the face and limbs on the side opposite to that of the motor or sensory tract which is implicated, or hemianopsia from involvement of the optic tract. Tumours in the region of the corpora quadrigemina may also cause dropsy of the ventricles, and the general (cerebral) symptoms which result therefrom.

Whether lesions (tumours) of the corpora quadrigemina produce

Fig. 58.—Photograph of Annie B.—Case of ophthalmoplegia externa acuta, showing the facial appearance at the height of the lesion.
The divergence of the eyeballs, the ptosis, the compensatory contraction of the occipito-frontales muscles, and the markedly stupid apathetic expression of countenance, are admirably shown.

any alterations of vision, except dimness or loss of vision, the result of double optic neuritis or post-neurotic atrophy (which may, as we have seen, be due to a tumour in any part of the intracranial cavity), or hemianopsia from pressure on the optic tract, seems doubtful.

Some observers think that the muscular inco-ordination and muscular spasms, which seem to result from lesions of the corpora

quadrigemina, are due to destruction or irritation of the subjacent tracts in the pons Varolii, more especially the superior cerebellar peduncles and tegmental tracts, rather than to the lesion of the corpora quadrigemina themselves. Ferrier, while admitting that it is impossible to separate the lamellæ of the corpora quadrigemina from the tegmental tracts with which they are in relation, states "that it is probable that lesions involving the subjacent tracts cause more marked and enduring disturbances of equilibrium; but that lesions

Fig. 59.—Photograph of Annie B.—Case of ophthalmoplegia externa acuta, showing the appearance on recovery.

This photograph was taken six months after that represented in Fig. 58. The expression is now bright and intelligent, and all paralytic symptoms have disappeared.

not directly involving these tracts are sufficient to induce marked disorders of equilibrium is, I consider, amply demonstrated by the actual facts of experiment."[1]

In a case which I have recorded with Mr. Berry, and in which the symptoms were, I think, probably due to a scrofulous lesion at

[1] *The Functions of the Brain*, Second Edition, p. 163.

the top of the pons (involving the nuclei of origin of the third nerve, and possibly also the corpora quadrigemina), ophthalmoplegia externa acuta was typically developed (see Figs. 58 and 59); there was marked general stupor (perhaps due to ventricular dropsy); and screaming fits, in which the jaws and hands were clenched, which were possibly similar to the screaming fits which Ferrier states are characteristic of electrical irritation of the testes of the corpora quadrigemina in the lower animals.

It is possible, but extremely unlikely, that a tumour at the top of the pons Varolii might involve individual parts of the third nerve

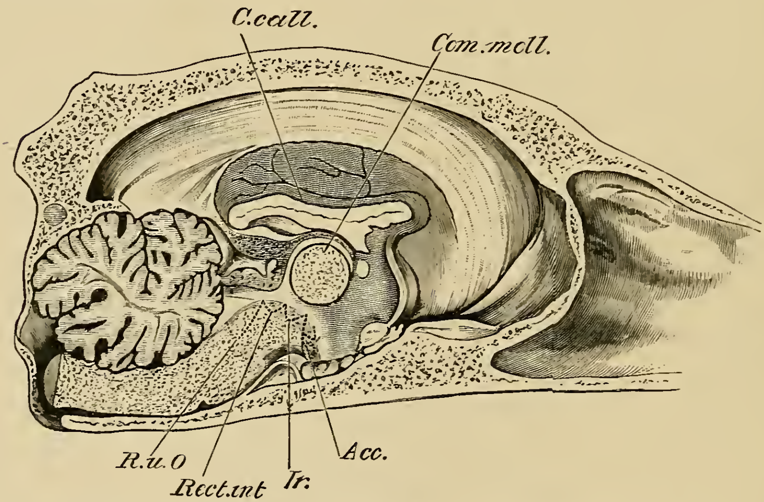

FIG. 60.—Sagittal section of the skull and encephalon of the dog (Hensen and Völckers).

C. call., corpus callosum ; *Com. moll.*, commissure mollis ; *Acc.*, centre of accommodation ; *Ir.*, centre for the sphincter iridis ; *Rect. int.*, centre for the rectus internus ; *R. u. O.*, centre for the rest of the ocular muscles.

nucleus, and so produce paralysis of accommodation only, abolition of the pupil reflex to light only, or paralysis of some of the individual muscles supplied by the third nerve.

The arrangement of the different component parts of the great nucleus of the third nerve at the top of the pons Varolii is extremely complicated and interesting. Thus Hensen and Völckers "have shown that the nuclei of origin of the third nerve contain separate centres for the individual oculo-motor actions. After removal of the cerebral hemispheres in dogs, they found that the application of

electrical stimulation to the floor of the aqueduct of Sylvius and posterior part of the third ventricle gave rise to different ocular movements, according to the position of the electrodes.

"Most anteriorly, in the wall of the third ventricle, is the centre for accommodation (Fig. 60, *Acc.*) acting on the tensor of the choroid through the anterior root fibres of the third nerve. Behind this is the centre for the constrictor fibres of the iris (Fig. 60, *Ir.*).

"Next, at the point of junction of the third ventricle with the aqueduct of Sylvius is the centre for the rectus internus (Fig. 60,

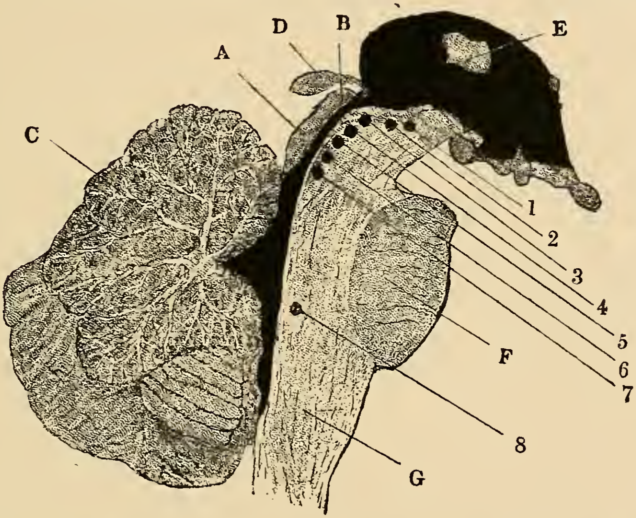

Fig. 61.—Longitudinal ventrical section through the human brain, showing (diagrammatically) the position of the nerve nuclei of the ocular muscles.

A, testes, and *B*, nates of corpora quadrigemina ; *C*, cerebellum ; *D*, pineal gland ; *E*, soft commissure in the middle of the third ventricle, which, with the aqueduct of Sylvius and fourth ventricle, are represented in black ; *F*, the protuberance of the pons ; *G*, medulla oblongata ; 1 to 6, different parts of the third nerve nucleus, viz.—1, centre for accommodation ; 2, centre for sphincter of the pupil ; 3, centre for internal rectus ; 4, centre for the rectus superior ; 5, centre for the levator palpebræ superioris ; 6, centre for the rectus superior ; 7, centre of the fourth nerve (trochlearis) for the superior oblique ; 8, centre of the sixth nerve for the external rectus.

Rect. int.), and further in order backwards, centres for the rectus superior, levator palpebræ superioris, rectus inferior, and lastly, that of the trochlearis or obliquus superior, the centre of which lies below the testes and somewhat laterally (Fig. 60, *R. u. O.*)."[1]

[1] Quoted from Ferrier's *Functions of the Brain*, Second Edition, p. 152. The original paper will be found in the *Archiv f. Ophthalmologie*, Bd. xxiv., 1878.

In Fig. 61 a longitudinal vertical section of the human brain is represented, the nerve nuclei being (diagrammatically) placed in the same position from before backwards as Hensen and Völckers have shown to be the case in the dog.

Kahler and Pick come to a slightly different conclusion as to the relative positions of the different nuclei. Whilst admitting (what indeed there is now abundant clinical experience to lead one to look upon as an established fact) that the centres for the nerve supply to the sphincter pupillæ and ciliary muscle lie detached from those for the fibres passing to the external muscles, also supplied by the third nerve, they were led from the post-mortem examination in two cases of incomplete nuclear paralysis of the third nerve (in one of which the elevators of the eye and lid were alone affected, and in the other, mainly the internal rectus), to locate the nuclei of the nerves to the elevator muscles in a line lying to the outer side of those destined for the supply of the internus and inferior rectus. This would bring the nuclei of such fibres as are physiologically associated into immediate proximity to each other—an arrangement which would seem more probable even *a priori* than that given in Hensen and Völckers' scheme.

Tumours of the pineal gland are probably more common than tumours of the corpora quadrigemina. The localising symptoms which they produce are very similar to those which result from lesions of the corpora quadrigemina, and are in fact due to involvement of these structures and of the adjacent parts (crus cerebri, pons Varolii, &c.), and to dropsy of the ventricles.

Tumours in the lateral half of the pons, below its upper third (*i.e.*, below the point at which the fibres of the facial muscles leave the main pyramidal tract to decussate in their passage to their nerve nucleus on the opposite side), may cause those forms of "alternate" hemiplegia in which (*a*) the arm and leg are paralysed on the opposite, and the muscles supplied by the facial nerve on the same side as the lesion (see Fig. 57, page 175); or (*b*) the arm and leg on the opposite and the face on both sides (see Fig. 62); or (*c*) the arm and leg on

the opposite side and the muscles supplied by the sixth or motor portions of the fifth nerve on the same side.[1]

Alternate hemi-anæsthesia, anæsthesia of the arm and leg on the opposite, and of the head and face, *i.e.*, the parts supplied by the fifth nerve on the same side; or anæsthesia of the arm and leg on the opposite, and of the head and face on both sides, may also be produced in the same manner.

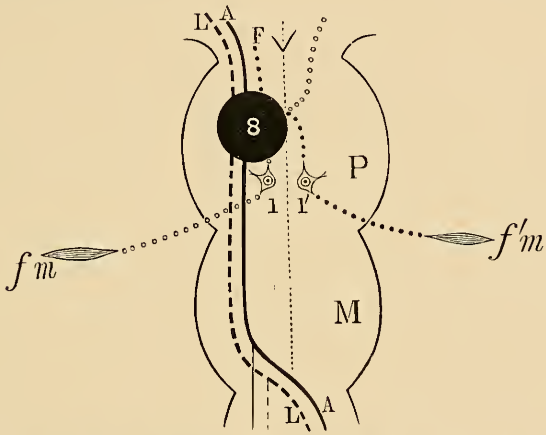

FIG. 62.—Diagram showing a lesion in the right half of the pons Varolii, causing paralysis of both sides of the face, and of the arm and leg on the opposite left side.

The letters *F*, *A*, *L* point to the motor tracts for the left side of the face, arm, and leg, as they enter the right side of the pons Varolii (*P*) in their passage from the right hemisphere of the brain. (Compare with Fig. 14, page 69.)

A lesion in the position of 8 (middle of the right side of the pons) may cause paralysis of the opposite arm and leg, and of both sides of the face (*f m* and *f′m*), by intercepting the motor fibres proceeding to the left side of the face (*f′m*), before they have decussated, and those passing to the right side of the face (*f m*) after they have decussated.

Should the root fibres or trunk of the fifth nerve be irritated rather than destroyed, neuralgic pains more or less violent in degree would be observed in the area of distribution of the affected nerve.

The sensory impairment, which results from lesions in the pons Varolii, is seldom so well marked or so definite in its distribution as the motor paralysis. An exception to this statement, however, occurs in those cases in which the tumour involves the sensory portion of the crusta, or the root fibres or trunk of the fifth nerve.

[1] *The Functions of the Brain*, Second Edition, p. 163.

Combinations of alternate hemiplegia and alternate hemi-anæsthesia, or of the different forms of alternate hemiplegia, may of course occur.

When the lesion (tumour) is so situated as to involve both sides of the pons and both pyramidal tracts, all four limbs may be paralysed; in such cases the loss of motor power is seldom complete or equally great on both sides of the body.

In rare cases, paralysis of one limb (monoplegia) is due to a lesion

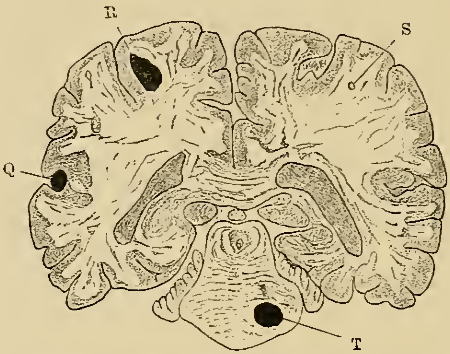

Fig. 63.—Transverse vertical section through the brain at the level of the greatest convexity of the pons Varolii, in a case of melanotic sarcoma, showing the position of melanotic nodules (*Q R S*) in the brain, and a large nodule (*T*) in the pons, involving the right pyramidal tract. (Reduced from a photograph.)

(tumour) in the pons; in one case of the kind, described by Dr. Allan Jamieson and myself in the *Edinburgh Medical Journal* for July 1887, page 40, and represented in Fig. 63, the paralysis became more extensive as the case progressed, and ultimately involved the face, arm, and leg, on the opposite side of the body.

A tumour in this situation may also involve the nerve nuclei or root fibres of nerves originating in the pons on one or both sides (see Fig. 64). A tumour originating in the neighbourhood of the middle line (median raphe) may involve the nerve nuclei of both sixth nerves and cause double internal strabismus. In cases of this description, the external rectus muscle on one side is usually paralysed before, or in a greater degree than, that on the other.

In some cases of tumour of the pons Varolii, vomiting is marked. In others, dementia and stupor are great, probably as the result of

copious ventricular dropsy, which is common in large tumours of the pons, and indeed in all large subtentorial new growths.

Deafness is not common, but it has been occasionally observed; it seems to be due either to direct pressure upon the auditory nerve, or, according to Ross, to the tumour being situated so as to involve the middle peduncle of the cerebellum. Ross states that he has seen "two cases" in which the tumour had extended from the middle

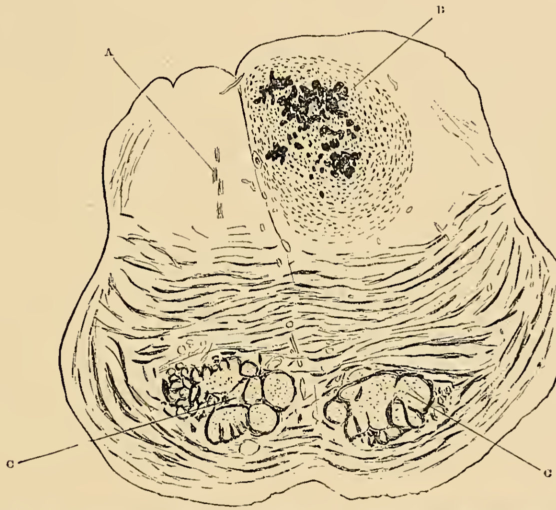

Fig. 64.—Camera lucida drawing of a transverse section through the pons Varolii, showing a small tubercular tumour in the upper surface of the left half. (Very slightly magnified — Hartnack, oc. 2, obj. 1, and drawing reduced from $8\frac{1}{2}$ to $2\frac{3}{4}$ inches.)

The section was stained with picro-carmine and mounted in Farrant's solution.

The letter *A* points to the fibres of the sixth nerve on the right side; they are not implicated. The letter *B* points to the tumour, which has destroyed the fibres of the sixth nerve on the left side. In the centre of the tumour there are several caseous masses. The letters *C C* point to the pyramidal tracts.

peduncle of the cerebellum to the pons, and in which unilateral deafness was an early and marked symptom.[1]

Disorders of equilibriation, or a tendency to fall to one side, &c., are present in some cases of tumour of the pons Varolii, and, in many cases appear to depend upon involvement of the middle peduncle of the cerebellum.

Conjugate deviation of the head and eyes may be due to a lesion

[1] *The Diseases of the Nervous System*, vol. ii., p. 591.

in the pons Varolii. It is, however, much more likely to be associated with acute lesions, such as hæmorrhagic extravasations, than with tumours.

The conjugate deviation of the head and eyes, which is due to a lesion of the pons Varolii, differs, as regards its direction, from the conjugate deviation of the head and eyes due to a lesion of the hemispheres. In lesions of one lateral half of the pons, the head and eyes are turned *away from* the side of the lesion (*i.e.*, towards the paralysed limbs) when the deviation is paralytic, and *towards* the lesion (*i.e.*, towards the side which is not convulsed—in other words, from the limbs which are convulsed) when the deviation is spasmodic.

In those cases, on the other hand, in which the lesion is situated in the cerebral hemisphere, the deviation is *towards* the lesion (*i.e.*, towards the sound side, or away from the paralysed limbs) when it is paralytic, and *away from* the lesion (*i.e.*, towards the limbs which are convulsed) when the deviation is due to spasm.

Difficulty in swallowing, paralysis of the tongue, paralytic difficulties of speech, vaso-motor disturbances, polyuria, glycosuria, and disturbances in the action of the heart or respiration, may also be caused by tumours in the pons Varolii. These are for the most part late phenomena, and are especially apt to occur when the medulla oblongata is pressed upon or otherwise involved by the lesion.

In some cases of tumour of the pons, sudden death occurs, apparently in consequence of paralysis of the respiratory or cardiac centres.

Tumours of the Medulla Oblongata.

Tumours originating within the medulla oblongata are rare; they are usually of small size, and are generally situated in the neighbourhood of the fourth ventricle. Headache is usually present; but, in the earlier stages, may be absent or slight. Double optic neuritis is perhaps more frequently absent than in tumours in other parts of the cranial cavity, possibly because of the small size of the tumour and the absence of any great increase of the intracranial pressure; but this point (the frequency of optic neuritis in the case of tumours of the medulla) requires further investigation. Vomiting is generally very prominent and urgent; hiccough is common.

In addition to hiccough and urgent vomiting, other localising symptoms, due to involvement of the eighth, ninth, tenth, eleventh, or twelfth nerves, or to the encroachment of the new growth upon the pons Varolii or cerebellum, together with paralysis of the limbs from pressure on, or involvement of, the pyramidal tracts in the medulla itself, may be present.

Bulbar symptoms—difficulty in articulation from paralysis of the lips, tongue, palate, or larynx—are specially characteristic and important. In some cases, there is glycosuria, polyuria, or albuminuria. The pulse frequency may be unduly slow. Cheyne-Stokes respiration is often present towards the termination of the case. Sudden death may occur from failure of the respiration, or perhaps from paralysis of the heart.

Paralysis of the limbs, which may be absent when the tumour is situated in the neighbourhood of the fourth ventricle, is usually present in a greater or less degree, when the tumour involves the anterior surface of the medulla, or presses upon the pyramids from without. Tumours which press upon the medulla from without are, as has been already pointed out, more apt to produce alternate hemiplegia, with paralysis of the tongue on the same side, than those new growths which are situated within the medulla itself.

Sensory disturbances (anæsthesia) are seldom prominent. Unsteadiness of gait, resembling, it is said, that due to cerebellar disease, has been met with in some cases, more especially when the tumour is situated in the neighbourhood of the fourth ventricle.

Tumours of the Cerebellum.

The cerebellum is a frequent seat of new growths, especially of scrofulous tumours. The symptoms, which are in many cases striking and characteristic, may be due partly to derangement of the function of the cerebellum itself, partly to pressure upon the pons Varolii and medulla oblongata, and partly to the increased intracranial pressure (and perhaps diffuse irritative changes throughout the brain) which results from extensive ventricular dropsy.

The headache in cases of cerebellar tumour may be very severe, and is often referred to the back of the head, but is not infrequently

frontal. In a small proportion of cases there is also tenderness on percussing the skull in the occipital region. *Vomiting* is very generally present. When very severe, it is probably, in some cases at all events, the result of irritation of the pons Varolii or medulla oblongata. Ferrier states that he never observed vomiting as a result of electrical stimulation of the cerebellum in the lower animals. *Double optic neuritis* is usually well marked, and is in many cases intense, associated with loss of vision, and followed by optic atrophy. *General mental impairment* (stupor, &c.) may be considerable, more especially in those cases in which the ventricular dropsy is great. *General convulsions* occasionally occur. In the case which is represented in Figs. 65 and 66 the patient died in, what was said to be, an epileptic fit.

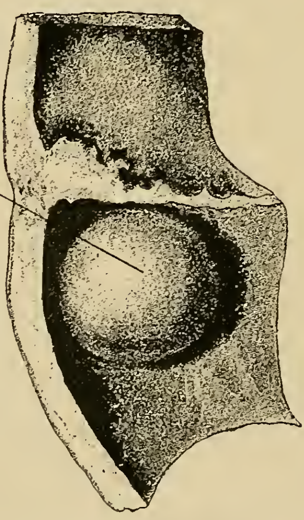

Fig. 65.—Portions of the temporal and occipital bones, showing a round tumour (*A*) about the size of a greengage plum, springing from the dura. The tumour was situated immediately below the tentorium, and had caused a deep indentation in the left lobe of the cerebellum.

In some cases (more especially in those in which the tumour is of small size, of slow growth, and situated in the lateral lobe of the cerebellum) there are no special symptoms indicative of the seat of the lesion. *An unsteady, irregular, reeling gait*, very like that due to alcoholic intoxication, is the most common and important localising symptom. The exact cause of the unsteadiness is a matter of debate. With Hughlings Jackson, I have been in the habit of regarding it as in some cases paralytic, and due to weakness in the muscles of the back; in others to inco-ordination in the same parts (muscles of the trunk). Ferrier thinks that it is not due to paralysis, but to an inability to co-ordinate the muscular adjustments required to maintain equilibrium or balance; that it is in fact a form, but a peculiar form, of ataxia (cerebellar ataxia).

Cerebellar ataxia differs in certain important particulars from the ataxia due to tabes dorsalis. When the patient stands with the legs

wide apart, the startings of the tendons, which are often present in locomotor ataxia, are not observed. The unsteadiness, too, is not markedly aggravated by closure of the eyes, and there are no disturbances of tactile sensibility, nor of the muscular sense. The irregular jerky movements of the legs which characterise the gait of locomotor ataxia are not present. Abolition of the knee jerk is in favour of locomotor ataxia; but it is not a certain guide to diagnosis, for it may also be lost in cerebellar disease.

In those cases of cerebellar disease in which the patient is blind from optic neuritis, or post-neurotic atrophy, the gait may of course

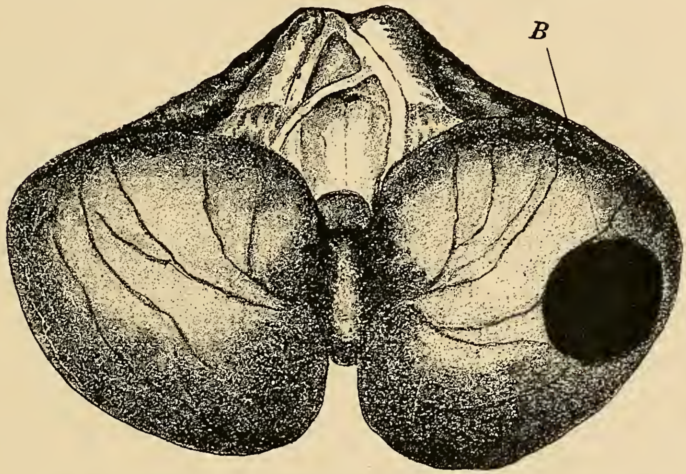

Fig. 66.—Cerebellum showing a deep indentation in its left lobe produced by the tumour shown in Figure 65. The cerebellar tissue was simply atrophied; there were no signs of inflammation or softening in the neighbourhood of the lesion.

The case is recorded in the *Edinburgh Medical Journal* for June 1879, page 1072.

be uncertain (like that of a man whose eyes are bandaged) independently of any true ataxia.

The unsteady, reeling gait is not present in all cases of tumour of the cerebellum. Small lesions, and even large lesions of slow growth, which produce destruction rather than irritation of the cerebellar tissue, may be compensated by voluntary efforts, *i.e.*, by the action of the higher (cerebral) centres. In such cases the unsteadiness of gait and difficulty in maintaining the equilibrium may be slight, or difficult to detect; but when the whole cerebellum has been involved

by disease, or has been found greatly or completely atrophied, careful observation has, says Ferrier, "never failed to discover a greater or less degree of awkwardness of movement and instability of equilibrium." [1]

The reeling gait seems to be most marked, and, according to some observers, only present, when the middle lobe is invaded. I have met with the most characteristic reeling gait in cases in which the tumour involved one lateral lobe or the middle peduncle, and in which the middle lobe was not directly involved, though it may have been pressed upon.

The disorder of equilibrium is greatest in those cases in which the lesion is unsymmetrical and unilateral; and it is probable that in man, as in the lower animals, the equilibrium is deranged in different ways in different cases, the exact character of the disorder depending upon (1) the exact part of the cerebellum which is involved, and (2) the manner in which it is affected, *i.e.*, whether irritated or destroyed.

The unsteadiness of gait is in some cases associated with, but is not the result of vertigo; for cerebellar ataxia may be present without any giddiness whatever. Marked vertigo is nevertheless suggestive of a cerebellar lesion. It is also probable that the character of the vertigo (the direction in which the patient thinks objects are moving past him, or the direction in which he feels as if he himself were being moved or whirled) varies with the exact part of the cerebellum which happens to be affected. It would appear that in different parts of the cerebellum there are separated and distinct centres, to which the nerves from the different semicircular canals of the internal ear and other peripheral end-organs concerned in the maintenance of balance proceed, and from which the necessary motor adjustments are distributed. Ferrier states that "the cerebellum would therefore seem to be a complex arrangement of individually differentiated centres, which in associated action regulate the various muscular adjustments necessary to maintain equilibrium and steadiness of the body, each tending to the displacement of the equilibrium round a horizontal, vertical, or intermediate axis acting as a stimulus to the special centre which calls into play the antagonistic or compensatory action. We should therefore expect to find that a lesion which annihilates the functional activity of any of the individual cerebellar

[1] *The Functions of the Brain*, Second Edition, p. 180.

centres should manifest itself in a tendency to the overthrow of the balance in the direction naturally opposed by this centre. This also is in accordance with the facts of experiment." And again—"Assuming," he says, "the seat of irritation to be correctly indicated by Crum Brown, we should regard the superior vertical canal as the afferent of the posterior cerebellar centres, the posterior vertical canal as the afferent of the anterior cerebellar centres, and the horizontal canal as the afferent of the corresponding lateral centres." [1]

In the case which is represented in Fig. 7, page 33, vertigo was a very prominent symptom, and the patient complained that when the eyes were open, the ceiling seemed to be falling down on the top of her.

Ferrier has shown that electrical irritation of the cerebellum in the monkey and other animals produces a series of movements of the eye, head, and trunk, and that the exact character of these movements depends upon the exact part of the cerebellum to which the irritation is applied. According to him, the following effects are produced by destruction and irritation of different parts of the cerebellum; and it seems probable, from what we know of cerebellar disease in man, that, in some cases at all events, somewhat similar phenomena may be noticed. Nystagmus is not uncommon in cases of cerebellar tumour.

Destruction of the anterior part of the middle lobe produces a tendency to fall forwards, while *irritation* of the same part excites the muscular combinations which would counteract this tendency, viz., backward movement of the head, extension of the trunk and limbs, together with upward movement of the eyes. These objective phenomena are probably accompanied by the subjective sensation of being revolved like a wheel on a horizontal axis from behind forwards.

Destruction of the posterior part of the middle lobe produces a tendency to fall backwards, while *irritation* of the same part calls into play the muscular adjustments necessary to counteract this tendency, viz., forward movements of the head and downward movement of the eyes. These objective phenomena are probably accompanied with the subjective sensation of being rotated like a wheel on a horizontal axis from before backwards.

Lesions of the lateral lobes or of the middle peduncle are, when complete, followed by rotatory movements, which in man would be round

[1] *The Functions of the Brain*, Second Edition, p. 199 *et seq.*

a vertical axis towards the sound side (*i.e.*, away from the lesion). After complete destruction of the lateral lobe or left middle peduncle, the patient would spin round and round from right to left. This rotation would probably be accompanied by the subjective sensation of vertigo, and of objects moving before the eyes from right to left.

Incomplete lesions of the lateral lobe or of the middle peduncle may, instead of causing a movement of rotation, cause a tendency to fall backwards and to the opposite side.

"The tendency," says Ferrier, "in simple and uncomplicated irritation seems to be overthrow of the balance towards the side of irritation, or in some axis inclining in this direction. . . . But the direction in which the balance is overthrown is not always towards the side of irritation, for I have observed it in several patients clearly towards the opposite side. This is probably due to over-compensation. The sense of falling to one side causes active volitional effort on the part of the individual, so that he falls in reality towards the opposite side. It is the presence of the hemispheres, and the intervention of conscious and volitional efforts, which complicate all the properly reflex phenomena of cerebellar adjustments. Hence it is that irritation of one side of the cerebellum causes the feeling of rotation towards or loss of support on the other side, because as the action which is called forth is in reality the adjustment to counteract such displacement, the two become indissolubly associated in consciousness, and the one effort invariably calls up the other. The feeling of loss of support on the opposite side of the body may be regarded as analogous to the apparent vanishing of objects in the same direction." [1]

As a matter of fact, a tendency to fall in some particular direction is frequently observed in cases of cerebellar disease in man. Vertigo, too, of a definite kind may also occur. Rotatory movements, with movements of the head and eyes, such as result from stimulation of different parts of the cerebellum in the lower animals, are much more rare, possibly because tumours of the cerebellum act more frequently as "destroying" than as "discharging" lesions, and the effects of slow destruction are compensated by voluntary effort.

Tumours of the middle lobe of the cerebellum are said to be attended, in about one-third of the cases, with symptoms of sexual

[1] *The Functions of the Brain*, Second Edition, p. 212.

excitement—increased sexual desire, and in males frequent erections with or without emissions. According to Bastian,[1] this genital excitement is perhaps due, not so much to disease in the cerebellar tissue, as to the irritation which such disease may set up in the posterior aspect of the bulb—a view which seems supported by the experimental observations of Ferrier, who never observed any sign of excitement of the generative organs in any of the animals experimented on.[2]

In addition to cerebellar ataxia, various other motor derangements may occur in cases of cerebellar tumour. Convulsive movements of a tonic kind, which may affect the muscles of the head and neck, trunk and limbs, and which produce in some cases retractions of the head, curving of the spine, and an appearance closely resembling a tetanic fit, are sometimes observed in cerebellar disease. In some cases these tonic convulsive movements are paroxysmal, in others the affected muscles are more or less continuously rigid, the head being retracted, or the trunk and limbs rigidly extended.

The exact significance of these tonic spasms and contractions is doubtful. Some authorities, such as Hughlings Jackson and Stephen Mackenzie, think that they are due to irritation and discharge of nerve tissue in the cerebellum itself. It is possible that in some cases (more especially where the convulsive movements resemble the movements which result from electrical irritation of the cerebellum in the lower animals) this is so. But I agree with those who think that the rigidity and tetanic-like spasms met with in cerebellar disease are in most cases due to the cerebellar lesion pressing upon, and producing irritation and discharge of grey matter in the pons Varolii or medulla oblongata.

Tremor, associated with voluntary movement, closely resembling the tremor of cerebro-spinal sclerosis, is sometimes seen, and is apparently caused by the pressure of the cerebellar tumour upon the pons Varolii or medulla oblongata, and the irregular transmission of motor nerve impulse, through the fibres of the pyramidal tracts, which results therefrom.

Paralysis of the limbs may also be produced in the same manner. When the pyramidal tract on one side is pressed upon, the arm and

[1] *Paralysis: Cerebral, Bulbar, and Spinal*, p. 348.

[2] *The Functions of the Brain*, Second Edition, p. 190.

leg on the opposite side are affected, for the pressure involves the fibres of the pyramidal tract above its decussation in the medulla. In this respect the paralysis which results from the pressure of a cerebellar tumour upon the pons Varolii or medulla oblongata, differs from the motor impairment associated with cerebellar ataxia, which is found on the same side as the cerebellar lesion. This is of course due to the fact that the middle peduncles of the cerebellum, which connect the lateral lobe of the cerebellum with the pyramidal tracts, decussate in the pons. The *right* lateral lobe of the cerebellum is consequently in this manner brought into relationship with the *left* pyramidal tract, as it lies in the pons; in other words, with the pyramidal tract proceeding to the right (the same side) of the body.

In some cases of cerebellar tumour, both pyramidal tracts are pressed upon, and the resulting paralysis involves both sides of the body, though not necessarily in the same degree.

The facial muscles are rarely paralysed unless the tumour presses directly upon the trunk of the facial nerve.

Deafness may be produced in the same manner (by direct pressure on the auditory nerve), but does not seem to be caused by lesions of the cerebellum itself. In one of my cases of cerebellar tumour, which has been previously referred to, total deafness was suddenly produced, apparently from sudden increase of the intracranial pressure.

Dimness of vision is often due to optic neuritis or optic atrophy, but is not directly caused by the cerebellar lesion.

Contraction of the pupils on the same side, and twitching of the ears, were observed by Ferrier to result from electrical irritation of the cerebellum; but whether any alterations in the size of the pupil result from cerebellar tumours in man, I do not know.

Enlargement of the head is not uncommon in young subjects, as the result of copious ventricular dropsy, and when it occurs is of some importance as a localising sign of tumours of the middle lobe, with which extensive ventricular dropsy is most frequently associated. The enlargement is bilateral, associated with opening up of the sutures, and exactly resembles the enlargement of ordinary chronic hydrocephalus.—(See Figs. 45 and 46, pages 149 and 150.)

Dr. Stephen Mackenzie thinks that the pressure upon the veins of

Galen is neither the sole nor the essential cause of dropsy of the ventricles. He says, "the hydrocephalus is usually ascribed to pressure on the veins of Galen, which, by increasing the lateral pressure on its radicles, causes exudation of their fluid contents, in the same way that pressure on the portal vein causes ascites. It will be readily seen from the diagram (Fig. 67) that a tumour of the cerebellum, especially of its middle lobe—a favourite situation—will be extremely likely to compress the venæ Galeni. But I believe that, whilst this may be a contributory factor in the production of the hydrocephalus, it is neither the sole nor essential one. Granting that a tumour pressing upon the veins of Galen causes dropsy of the

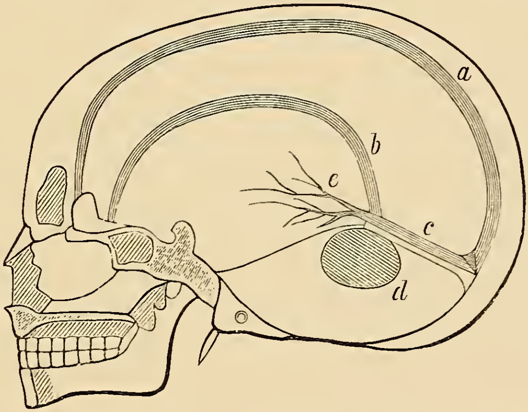

Fig. 67.—Diagram showing the manner in which compression of the veins of Galen is produced by a tumour of the cerebellum, more especially of its middle lobe. (After Stephen Mackenzie.)

The letter *a* points to the superior, and *b* to the inferior longitudinal sinus; *c* to the straight sinus; *d* to a tumour beneath the tentorium; and *e* to the veins of Galen.

ventricles, as long as the outlet of the general ventrical cavity remains patent, as the fluid collects it will flow away; moreover, if the pressure be extreme, it will cause obliteration of the vein. But it will be readily seen by reference to the diagram (Fig. 68) that subtentorial tumours, especially when involving the middle lobe, will be extremely likely to cause constriction or obliteration of the cerebro-spinal foramen, or some point above this—the fourth ventricle or aqueduct of Sylvius. . . . When the communication between the general ventricular cavity and the subarachnoid space is obliterated, the fluid

poured out by the choroid plexuses, and possibly the obstructed venæ Galeni, is dammed up, distends the ventricles, and causes the extreme hydrocephalus sometimes found. . . . The effects of this dropsy of the ventricles are very interesting and characteristic. The very gradual and equable (water) pressure to which all parts of the brain are subjected causes a gradual 'blotting out' or dissolution of what mental faculties have been obtained, and the order of this blotting out is very instructive to the psychologist. From a conscious volitional being, the patient is reduced to a mere automaton, or to a vegetative existence. In this condition he may continue for a considerable time."[1]

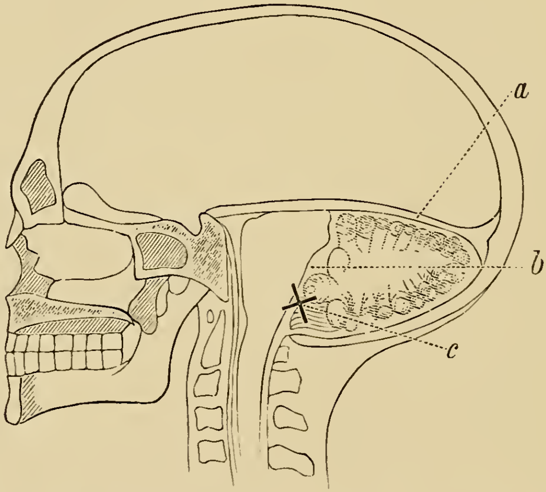

FIG. 68.—Diagram showing the manner in which tumours of the cerebellum interrupt the communication between the cavities of the ventricles and the subarachnoid space. (After Stephen Mackenzie.)

The letter *a* points to the tentorium cerebelli; *b* to the fourth ventricle; *c* to the cerebro-spinal foramen.

A tumour in the situation of the cross would obliterate the cerebro-spinal foramen; while a tumour a little above the point marked by the cross might obliterate the cavity of the fourth ventricle.

In tubercular cases, and indeed in other forms of new growth, when the case is of long duration, there may be considerable emaciation. In many, but by no means in all cases of tubercular tumour of the cerebellum, there is evidence of tubercle in the form of enlarged glands, diseased joints, diseased lungs, &c., in some other

[1] *Lancet*, vol. i., 1880, p. 559.

part of the body. In some cases in which there is no actual manifestation of tubercle, the hereditary history is so strong as to suggest that the cerebellar lesion is scrofulous.

The Differential Diagnosis of Cerebellar Tumour and Ménière's Disease.

In both conditions, vertigo and derangements of co-ordination may be marked symptoms, and the character of the vertiginous sensations and of the ataxia may be exactly the same. The cerebellum would indeed appear to be the centre to which the labyrinthine nerves pass, and the disturbances which result from irritation or destruction of the end-organ (semicircular canals) are necessarily therefore identical with those due to lesions of the cerebellar centres themselves. In both conditions there may be vomiting, and in both convulsive twitchings.

Though in these respects the two conditions closely resemble one another, the distinction can as a rule be readily made; for in the one there are usually symptoms and signs of a *central*, and in the other of a *peripheral* lesion.

In *cerebellar tumour*, marked headache and double optic neuritis are distinctive symptoms, and there may be evidences of motor paralysis from pressure on the pons Varolii or medulla oblongata. Deafness and facial paralysis are seldom present.

In *Ménière's disease* there is no marked headache, no optic neuritis, and no paralysis of the limbs, unless the condition is associated with meningitis or some other cerebral complication, such as abscess, which may cause these symptoms. While signs of local disease of the ear (purulent discharge, &c.) are often present, there is usually deafness or some other alteration of hearing; and there is often peripheral facial paralysis, due to involvement of the facial nerve in the Fallopian canal.

The Differential Diagnosis of Cerebellar Tumour and Disseminated Cerebro-Spinal Sclerosis.

The diagnosis is usually easy, but in some cases most difficult. In cerebellar tumour, headache is usually much more severe than in

cerebro-spinal sclerosis. Vomiting, which is so often such a common symptom in cerebellar tumour, is seldom observed in cerebro-spinal sclerosis. Double optic neuritis is usually present in cerebellar tumour, but is very rarely, if ever, met with in cerebro-spinal sclerosis. In the latter affection I have, however, noticed well-marked, and in one case apparently post-neurotic, optic atrophy.

The characteristic tremor is usually much greater, and is developed at a much earlier stage in cerebro-spinal sclerosis. It is chiefly in the later stages of cerebellar tumour that the pressure on the pons Varolii becomes so considerable as to produce this symptom.

Nystagmus is more common in cerebro-spinal sclerosis. The peculiar vacant look, and the characteristic scanning speech of cerebro-spinal sclerosis, have not, so far as I know, been noticed in cerebellar tumour.

A careful consideration of the whole progress and course of the case, together with the special symptoms in each individual instance, will usually enable the observer to differentiate the two conditions without much difficulty.

CHAPTER IX.

PATHOLOGICAL DIAGNOSIS AND MORBID ANATOMY.

The determination of the pathological nature of the tumour is in many cases a matter of great importance, both for prognosis and treatment; for, although in the present position of science the syphilitic is the only form of intracranial tumour which can with any certainty be influenced by treatment, it is possible that, in the future, we may be able to restrain the growth or to produce the absorption and complete cure of intracranial tumours by means of internal remedies (drugs).[1] And at the present time the determination of the pathological nature of the tumour is of great importance from the operative point of view. No one, for example, would recommend an operation for the removal of an intracranial melanotic sarcoma, or of any other multiple tumour, such as secondary cancer. The removal of a gliomatous tumour, too, is in some cases questionable, on account of the diffuse and extensive infiltration which is often present in these cases, and the consequent impossibility of completely removing such new growths. On the other hand, where there is reason to suppose that the tumour is encapsuled (such a form of new growth, for example, as an encapsuled sarcoma), an operation is indicated, provided that the other conditions for active interference (see page 246) are favourable.

The most common forms of new growths, which are found within the cavity of the cranium, are scrofulous and syphilitic tumours; next in order of frequency come gliomata, and the various forms of sarcoma; then, but a long way behind, cancers and endotheliomata; lastly, cysts of various forms (simple and parasitic); psammomata, cholesteatomata, neuromata, fibromata, osteomata, lipomata, melanomata, &c.

[1] Scrofulous tumours are in some instances materially benefited by appropriate treatment (see p. 248); and in some cases the active growth of sarcomata is apparently restrained by the internal administration of arsenic.

Scrofulous tumours are the most common form in young people, *i.e.*, below the age of fifteen, and syphilitic tumours in adults between the ages of twenty-five and fifty.

In some cases the tumour is primary, in others secondary. The new growth may originate in the connective tissue of the brain or its membranes, in the bones of the skull, in the pituitary body or pineal gland; but, with perhaps the single exception of the rare form which is termed a neuroma, the tumour does not grow from the nervous tissues proper.

Secondary tumours (deposits) may reach the brain either through the blood-vessels or lymphatics, and, for the reasons previously given (see page 3), the primary new growth is frequently pulmonary.

Various degenerations (such as the myxomatous, caseous, and calcareous) may affect intracranial like other forms of new growth.

The determination of the pathological diagnosis is in some cases easy; in others most difficult or impossible. Indeed, in many cases the pathological diagnosis is rather a matter of probability or of numerical chance, than of logical conclusion based on definite facts.

In trying to arrive at a pathological diagnosis the following are the most important points to which attention should be directed:—

(1.) *A history of the removal of a previous new growth of known pathological character, or the presence of associated new growths (of known pathological character) in some other part of the body.*

When symptoms of a cerebral tumour develop some months after the removal of a pigmented mole or melanotic sarcoma, the diagnosis, both that there is a tumour, and that it also is a melanotic sarcoma, is of course self-evident. So again, when symptoms of a cerebral tumour develop, and a sarcomatous tumour is present on the ribs or sternum, or when there is evidence of cancer in the liver or a history of the removal of a cancerous breast, the pathological diagnosis does not require to be sought for, for it, as it were, declares itself.

(2.) *The presence of symptoms or signs of some dyscrasia, diathesis, or special constitutional condition which predisposes to a particular form of new growth.*

The presence of other distinctly syphilitic lesions (such as nodes on the exterior of the skull or shins), the evidences of former syphilitic lesions (cicatrices of previous ulcerations), or the presence

of a marked dyscrasia (such as the muddy, unhealthy complexion which is so common in constitutional syphilis), would of course be strongly in favour of the (cerebral) tumour being syphilitic. So, too, the presence of enlarged and caseous glands, of scrofulous joint disease, of phthisis, or other distinct tubercular manifestations, would be strongly suggestive of the scrofulous character of the new growth.

Again, a strong hereditary history of scrofula in a child, or of cancer in an old adult, would suggest that in the former the cerebral tumour was scrofulous, and in the latter cancerous. The diagnostic value of an hereditary tendency to a particular form of new growth is seldom, however, very great.

(3.) *The age of the patient.*—This is of some little value. Scrofulous tumours are by far the most common form of new growth in children, Syphilitic tumours are practically never seen in children, but almost always in adults; for they are rarely, if ever, the result of inherited, but are almost always due to acquired syphilis. Gliomata may occur at any age, and so may sarcomata. Cancers are almost always seen in old people.

(4.) *Sex.*—This is of little value. Most forms of cerebral tumour, but more especially syphilitic growths, are more common in males; but the fact is seldom of much value to the diagnostician.

(5.) *A history of injury.*—The fact that the tumour has developed immediately after and is apparently the result of an injury, is, I think, suggestive that it is either a syphiloma, a sarcoma, a glioma, or a scrofulous tumour.

(6.) *The situation of the new growth.*—This is of considerable diagnostic value. The most frequent seat of scrofulous tumours is the cerebellum. Tumours of the pons Varolii are usually either scrofulous, gliomatous, or syphilitic. Tumours on the surface of the hemispheres are usually either syphilitic, sarcomatous, or localised tubercular deposits. Primary tumours of the centrum ovale and corpus callosum are usually gliomata or sarcomata. Tumours at the base are usually enlargements or new growths of the pituitary body, aneurisms of the large vessels, syphilitic gummata, cancers, or sarcomata.

(7.) *The style of the symptoms, and the manner of their development, the duration of the case, and the effects of special modes of treatment.*—These are all of some diagnostic importance.

The presence of symptoms indicative of several separate lesions is suggestive of syphilis, or of some secondary form of tumour (scrofulous, melanotic sarcoma, cancer, &c.).

Jacksonian epilepsy is suggestive of a cortical lesion, and more especially of syphilis or tubercle.

Fig. 69.—Enlargement of the head in a case of so-called perforating tumour of the dura mater. (After Drummond.)

Pseudo-apoplectic attacks are suggestive of syphilis or glioma. True apoplectic attacks, followed by lasting paralysis, are suggestive of hæmorrhage from a glioma, but they may be due to syphilitic disease and plugging of a large vessel.

Perforation of the bones of the skull (such as is represented in Figs. 69, 70, and 71) only occurs, so far as I know, in sarcomata and cancers.

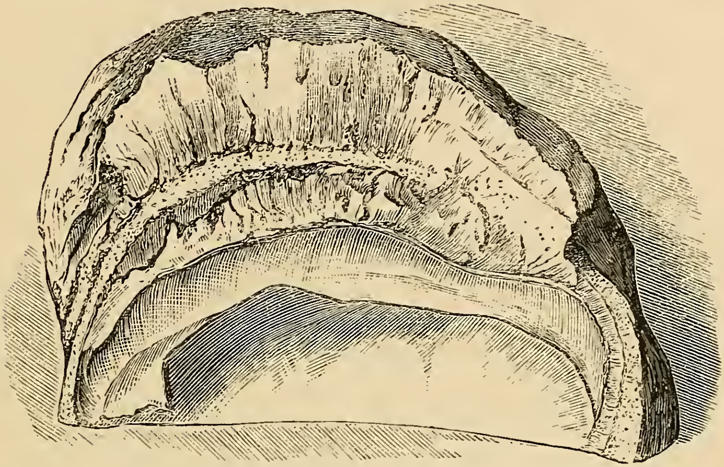

Fig. 70.—A section through the left and smaller half of the skull, and new growth in the case of perforating tumour of the skull, represented in Fig. 69. (After Drummond.)
The section was made just to the right of the falx; the right hand side of the figure corresponds to the frontal end.

The case illustrated in Figs. 69 and 70 is reported by Dr. D. Drummond in the *British Medical Journal*, October 20, 1883, page 762.

The patient, a boy, aged five, was admitted to the Children's Hospital, Newcastle-on-Tyne, on December 5, 1882, under the care of Dr. Baumgartner, suffering from headache, vomiting, and double optic neuritis. There was also a swelling on the right side of the head near the vertex. This swelling rapidly increased in size until the head measured at the point of greatest circumference above the ears twenty-six inches, and presented the appearance shown in Fig. 69. The right eye became involved and destroyed by the new growth; other tumours developed in the lower jaw and testicle.

The patient died on March 21, 1883. At the post-mortem examination the tumour was removed with the brain and most of the cranium, leaving the scalp attached to a portion of the base of the skull, including the face. The tumour mass covered the right frontal, right parietal, and part of the occipital bones, and extended over the middle line to the left side for about four inches, so as to involve a considerable portion of the left parietal and frontal bones. The right frontal, and portions of the right parietal bone, were much thickened, and in places so softened and infiltrated with the new growth as to admit of the ready passage of the knife through the tumour into the brain beneath. The surface of the mass, after the skin was removed, presented a peculiar piebald appearance, being principally of a deep red colour, with patches of yellow and pink. The right frontal, parietal, and temporo-sphenoidal lobes of the brain were much compressed, and masses of soft, red, cancer-like new growths were attached to the cortex of the upper and posterior part of the first temporo-sphenoidal convolution, the supra-marginal and angular gyri, and part of the occipital lobe. On making a longitudinal section through the tumour and skull, the structure, which was soft and elastic, was seen to be of a deep red colour, presenting a highly hæmorrhagic appearance, especially the portion between the dura mater and the inner surface of the cranial vault.

The whole thickness of the tumour was about two inches, of which nearly three-quarters of an inch lay beneath the skull. The tumour was firmly adherent to the external surface of the dura mater, which it pierced in places. Here and there the tumour-mass was studded with long spiculæ of bone; or, to be more accurate, the tumour tissue had invaded the bones, expanding, softening, and at the same time thoroughly infiltrating them.

On microscopical examination the tumour was found to be a highly vascular, small

In cases of cerebral syphilis, the symptoms usually develop quickly, and often subside rapidly under large doses of iodide of potassium and mercury.

Gliomatous tumours are often of very long duration, and so are many encapsuled sarcomatous tumours growing from the membranes.

FIG. 71.—Perforating tumour of the dura mater. (After Drummond.) The case is reported in the *British Medical Journal*, October 20, 1883, page 763.

In such cases the general symptoms may be characteristic of tumour, but localising symptoms are often wanting.

Some other points of a similar nature might be mentioned, but they will perhaps be better described in connection with the special features of each form of new growth, to which we must now proceed.

THE SCROFULOUS TUMOUR.

Tubercular tumours are the most common form of new growth in young people. The cerebellum is the most frequent seat, but the pons

Varolii and cerebrum (both cortex and basal ganglia) are frequently affected. In some cases the tumour is single, in others multiple. I have seen as many as eight separate scrofulous nodules in different parts of the same brain.

FIG. 72.—Right hand of a patient suffering from ophthalmoplegia externa acuta, which was probably due to a tubercular lesion at the top of the pons Varolii, showing scrofulous enlargement of the middle finger. (Copied from a photograph.)

Other tubercular lesions, such as enlarged caseous glands, scrofulous disease of the bones or joints (see Fig. 72), phthisis, &c., are very generally present, but are not always detectable before death. In all cases of scrofulous brain tumour there is probably some source of local infection; a very common one, which is often unsuspected during life, is a caseous mesenteric gland.

In many cases tubercular meningitis is developed in association with scrofulous tumours of the brain or cerebellum; and the clinical picture may for this reason be complicated.

The scrofulous tumour varies in size from the smallest possible nodule up to a large mass as big as a turkey's egg. The form of the tumour is generally more or less rounded; the consistency is usually hard, though there may be softening, or even the formation of a cavity filled with broken-down *débris* in the centre of the mass. The colour is yellow or rather greenish-yellow. The tumour is developed in the substance of the nerve tissue, and in this respect

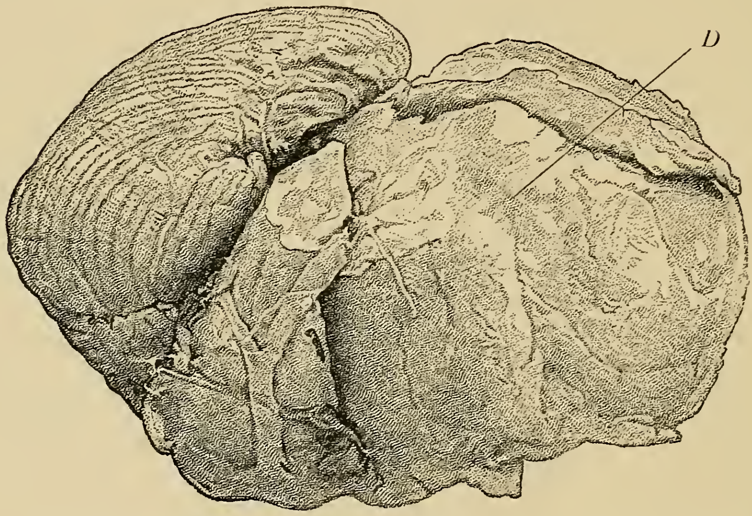

Fig. 73.—Large scrofulous tumour of the right lobe of the cerebellum. Copied from a photograph (reduced in size).

The letter *D* points to the tumour, to the outer surface of which the dura mater is adherent (the line passing from the letter *D* to the tumour crosses the dura, which has been partly turned back off the tumour).

The left lobe of the cerebellum is natural, and forms a remarkable contrast as regards size to the corresponding part on the opposite side.

presents a marked difference from most syphilitic tumours, which may easily enough be confounded with it. The nervous tissue around the tumour is usually softened or inflamed. The membranes over the surface of the tumour are in many instances adherent to the affected portion of brain tissue (see Fig. 73, in which the dura mater is firmly adherent to the left lobe of the cerebellum, in which a large scrofulous tumour is situated). When the scrofulous tumour reaches the surface, it may extend by direct contiguity from one portion of the brain to another. I have seen a scrofulous tumour extend in this way directly from the surface of the left, to the surface,

and thence to the interior, of the right lobe of the cerebellum. In some cases the tumour becomes calcified.

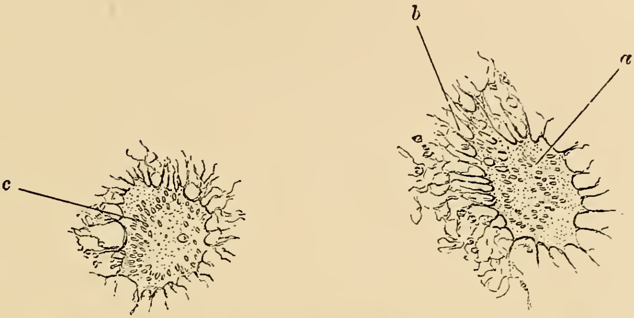

FIG. 74.—Giant cells from the scrofulous tumour of the pons Varolii, shown in Fig. 64, page 185. H., oc. 3, obj. 8, tube out, and drawing reduced one-third.

FIG. 75.—Camera lucida drawing of a transverse section through a portion of the scrofulous tumour of the pons Varolii, shown in Fig. 64. Stained with picro-carmine; magnified, oc. 3, obj. 8, tube out; and drawing reduced from $4\frac{1}{2}$ to 3 inches.

The letter *B* points to a dense cellular infiltration in the coats of the blood-vessel; *A*, to a similar infiltration in the surrounding cellular tissue; *D*, to large cells in the surrounding tissue; and *C*, to the junction of the tumour with the normal tissue of the pons.

On microscopical examination the usual characteristics of a scrofulous lesion are found (giant cells, and, with satisfactory methods of

preparation, tubercle bacilli). Fagge thinks that the cells which surround scrofulous tumours of the brain are larger than the cells found in tubercle in other organs. The microscopic appearances characteristic of the scrofulous tumour are well seen in Figs. 74 and 75.

The diagnostic features of the scrofulous tumour are often sufficiently characteristic to enable a positive opinion as to the nature of the tumour to be given.

By far the most important fact is the presence of tubercle in some other part of the body, or cicatrices in the neck or other evidences of former scrofula. Failing positive evidence of this kind, the peculiar build of the thorax and body generally, which we know from experience is apt to be associated with phthisis; a strong hereditary history of scrofula; the fact that the patient is a child, or at all events young; that the tumour is situated in the cerebellum; that the general health is (in some cases at all events) considerably affected; and that there is no evidence to show that the tumour is syphilitic, sarcomatous, &c., are the most important points. It should further be remembered that in some cases a head injury seems to be the exciting cause of this form of new growth.

The Syphilitic Tumour.

This is the most common form of new growth in adults (between the age of twenty-five and fifty), but is very seldom seen in younger persons or children; for it is almost always the result of acquired syphilis, and is very rarely indeed due to the inherited form of the disease. Dr. Wood has "seen cerebral syphilis occur at twenty years of age as the first evident outbreak of inherited syphilis;" but this is quite exceptional.[1]

In males, a history of primary syphilis can usually be elicited. It is important to remember that in many cases in which cerebral syphilis subsequently develops, the secondary symptoms are slight,—in fact, they may be so insignificant as to escape attention. The length of time which elapses between the primary chancre and the development of cerebral symptoms is very variable, but is usually several years. Cases have, however, been recorded in which cerebral

[1] Pepper's *System of Medicine*, vol. v., p. 1007.

symptoms seem to have developed within a few months of infection. In some cases, injury to the head appears to be the exciting cause of this form of tumour.

The intracranial lesions which may result from syphilis are numerous. The most important are—(1.) Intracranial nodes, with or without meningitis. (2.) Gummata. (3.) Syphilitic lesions of the large or medium-sized vessels (chiefly endarteritis obliterans), the result of which is diminished or arrested blood supply to well-defined, and in some cases extensive, areas of the brain tissue, with the consequent production of well-defined, naked-eye lesions, such as softenings. (4.) Localised inflammation (cerebritis) or softening in the neighbourhood of gummata and meningeal lesions. (5.) Inflammation of the intracranial peripheral nerves. In cases of this kind, the inflamed nerve is usually infiltrated by a gummatous deposit; but it is probable that syphilitic neuritis may be present without gumma or meningitis. (6.) Meningeal inflammations without gummatous deposits. Whether syphilis does produce a more or less generalised inflammation of the soft membranes, which may result in adhesion of those membranes to the surface of the brain, without localised gummatous deposits or other well-marked syphilitic lesions, is doubtful. (7.) Disease of the minute vessels (in many instances periarteritis, I believe, rather than endarteritis obliterans), with resulting minute changes in the cerebral cortex—atrophy or slow destruction of the nerve elements, increase of the connective tissue, and a final condition of sclerosis and atrophy of the grey matter.

Now in most cases of intracranial syphilis, although one form of lesion may predominate, or appear to predominate, the other, or some of the other forms of lesion described above are usually present in some degree. Possibly any one of these lesions may be present alone or in combination with any other.

The form of cerebral syphilis with which we are here specially concerned is the syphilitic tumour or gumma. In the vast majority of cases it is situated on the surface of the brain (see Figs. 76, 77, and 82), very often in the frontal or Rolandic areas, or at the base. I have, however, met with two cases in which a syphilitic gumma was situated in the substance of the brain (see Fig. 79).

More or less meningitis is almost always found in the immediate

neighbourhood of the gumma, and in many cases the meningeal inflammation is the most striking feature of the case (see Fig. 76).

The lesion gradually invades the brain substance, and this process of invasion, which is well represented in Figs. 77, 78, and 80, is attended both clinically (during life) and microscopically (after death) with striking evidences of irritation.

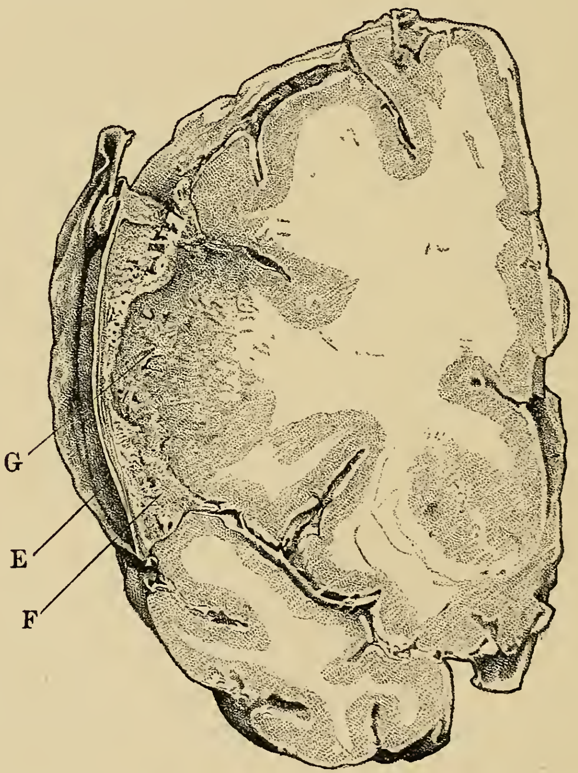

Fig. 76.—Transverse vertical section through the left hemisphere of the brain (frontal region and tip of the temporo-sphenoidal lobe) in a case of gummatous syphilis. (Seen from behind.)

The dura, to which the letter *E* points, is thickened and adherent; the letter *F* points to the gummatous and inflammatory products situated between the membranes and the surface of the brain; the letter *G* to the surface of the brain, at a point where it is being invaded by the syphilitic lesion.

Another most important feature of the syphilitic gumma, from a clinical point of view, is the fact that the nerve trunks and vessels in its neighbourhood are apt to be involved and infiltrated by the lesion (see Figs. 82 and 86).

In many cases there are several syphilitic lesions in the same brain—see Figs. 81, 82, and 83, in which an enormous softening, due

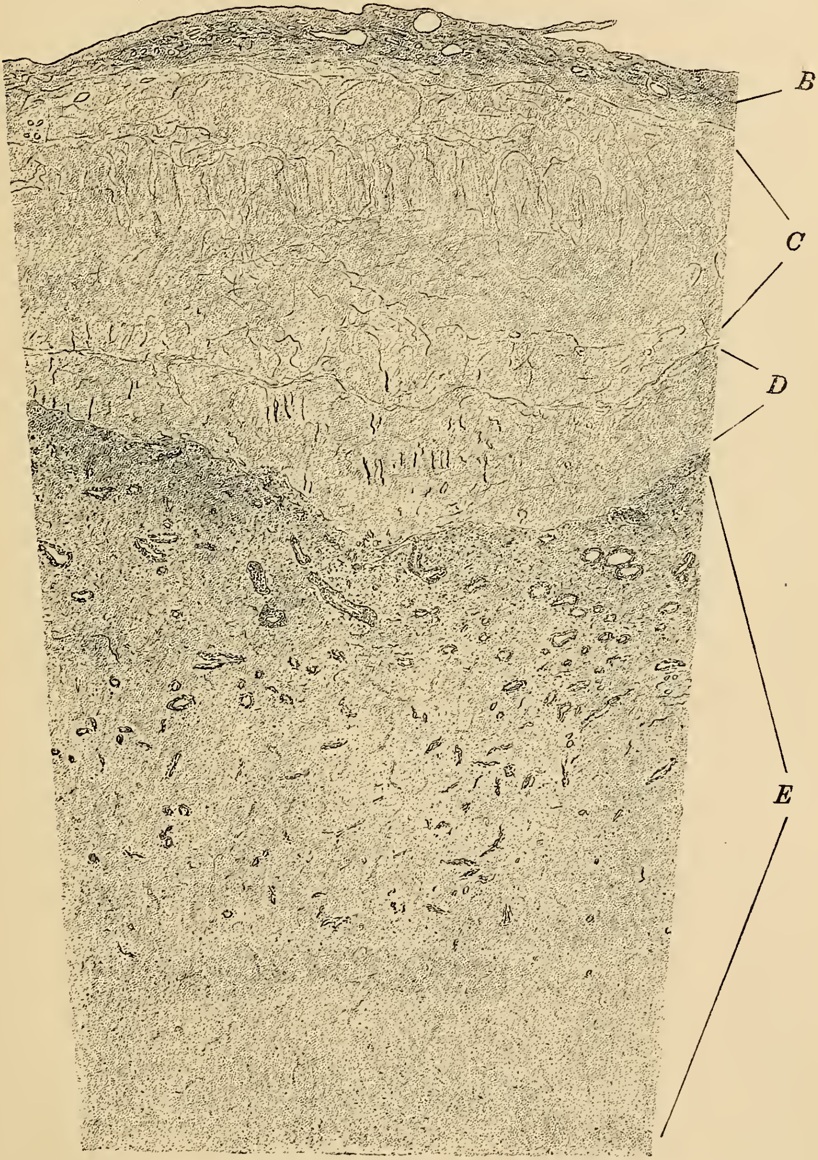

Fig. 77.—Camera lucida drawing of a microscopical section through the membranes and cortex of the brain in a case of syphilis; showing thickening of the membranes (*B* and *D*), with gummatous products (*C*) between them; and invasion of the superficial portion of the cortex cerebri (*E*). Low power—Hartnack, oc. 3, obj. 1; and drawing reduced from $7\frac{1}{2}$ to $6\frac{1}{2}$ inches.

to syphilitic arterial disease, was associated with a well-marked gumma, meningitis, and involvement of the third, optic, and other nerves.

The naked-eye appearances which the syphilitic gumma presents,

FIG. 78.—Camera lucida drawing of a portion of a microscopical section through the deeper layers of the greatly thickened pia mater (*A*) and the superficial portion of the cortex cerebri in a case of syphilitic disease. Hartnack, oc. 3, obj. 4 ; drawing reduced from $5\frac{3}{4}$ to 4 inches.

B, junction of pia and cortex ; *C*, diseased, and *E*, comparatively healthy cortex ; *D*, diseased vessel.

cannot be better described than in the words of Heubner. He says, " The most frequent change met with in the cranium of patients, who develop cerebral disease under the influence of syphilitic cachexia, is the formation of a heteroplastic tissue, the syphiloma or gummous tumour. We shall therefore describe it first. The new growth appears in two very different forms, which, however, are often united in the same body, either (first) as a whitish or greyish-red or moist grey mass, of the consistency of a firm jelly, in thinner places half trans-

lucent; of irregular form, generally determined by the place where it is seated; discharging upon its cut surface a scanty whitish-red juice, and gradually blending with the surrounding tissues; or (second) as

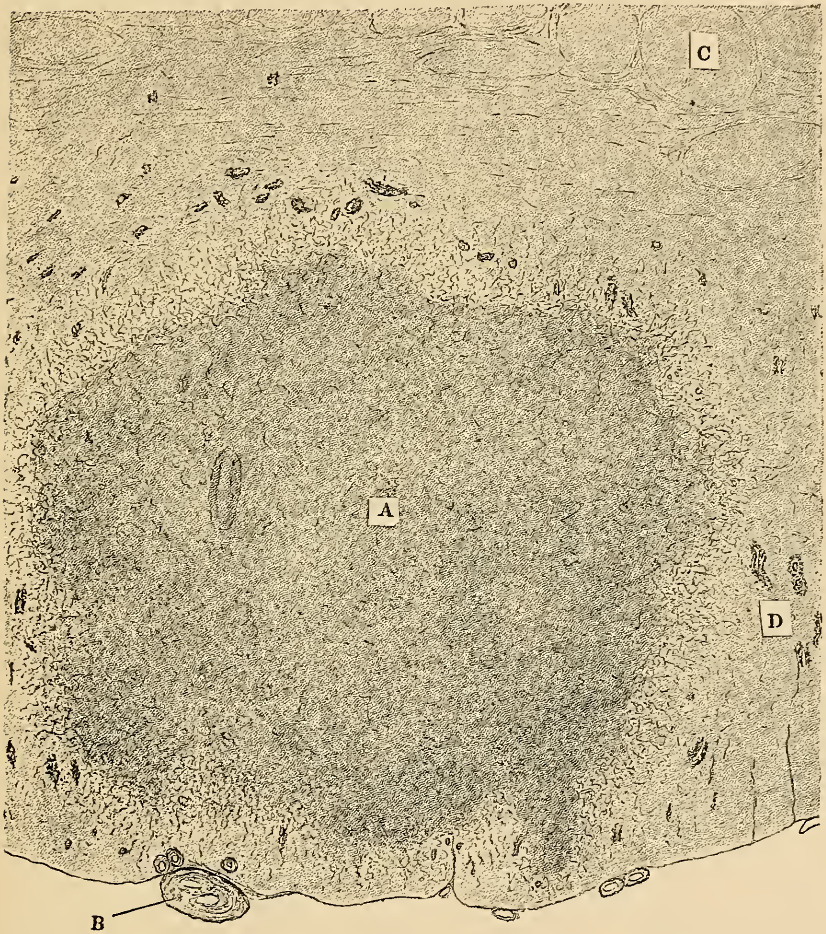

FIG. 79.—Camera lucida drawing of a microscopical section through the pons Varolii, showing a syphilitic gumma (*A*) in its interior. Hartnack, oc. 2, obj. 1; and drawing reduced from $6\frac{1}{3}$ to $4\frac{3}{4}$ inches.

A, gumma; *B*, diseased vessel on surface of pons; *C*, bundles of the pyramidal tract; *D*, substance of pons surrounding gumma. There is, it will be observed, no meningitis.

a yellow, firmer—often as firm as cartilage—dry, friable substance, upon section homogeneous and cheesy, which is found either in the form of somewhat sharply circumscribed larger or smaller tumours, or

invading the greyish-red substance just described, sometimes as larger nodules and streaks, and sometimes as a more minute marbling.

"The greyish-red mass consists chiefly of round cells, with a small proportion of nuclei, and, among them, spindle and stellate cells, arranged sometimes apparently without order, and sometimes forming groups together with an alveolar framework. These cell masses lie, as far as possible, in the interstices of the original tissue, the constituents of which, altered or not, form the chief part of the intercellular substance of the new growth, which is permeated by scanty capillaries,

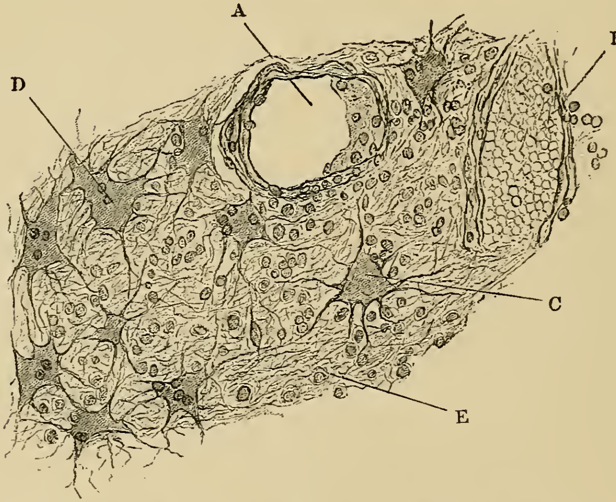

FIG. 80.—Camera lucida drawing of a microscopical section through a syphilitic gumma of the brain, showing enlarged connective tissue cells (*C* and *D*), and infiltration of the intervening tissue with corpuscular elements (*E*). Hartnack, oc. 3, obj. 8, tube out; and drawing reduced from 4 to 3 inches.

A transversely, and *B* longitudinally divided vessels.

often of large calibre. Hence in places where the original tissue was delicate and contained much water, the growth is softer (subarachnoid space); where it was more dense and fibrous, firmer (dura mater). Where the interstices of the tissue are large, there numerous cells lie together in large alveolar spaces, which may be again divided into secondary spaces by finer fibres (Wagner); where the meshes are small, only a few cells lie in narrow fissures. In the first case we have to do with a growth rich in cells; in the latter, with one more fibrous and brawny. The source of the chief mass of the cells is

probably to be found in the blood; the granulation cells have mostly emigrated; the spindle cells, which in many places are arranged in a more organised structure, as young connective tissue, in the meshes of which round cells lie, probably arise from the connective tissue strata of the place at which the growth originated. One can often observe how the same growth, which in one place has the character of indifferent granulation tissue (for instance, in the subarachnoid space), upon reaching another tissue (for instance, brain substance), immediately assumes an exquisite alveolar structure. In tumours of

Fig. 81.—Transverse vertical section through the frontal lobes of the brain, just in front of the tip of the temporo-sphenoidal lobe, in a case of cerebral syphilis; showing an extensive softening of the right hemisphere, the result of syphilitic disease of the nutrient vessels. Facsimile representation of a photograph (reduced in size).

The softening, it will be noticed, extends for some little distance across the corpus callosum.

the subarachnoid space, the mass of cells lies embedded in a gelatinous intercellular substance within the meshes of connective tissue.

"The whole new-formed tissue is permeated by blood-vessels, chiefly of a capillary character, which are more or less abundant according to their abundance in the original structure; in some places little extravasations may be found, which mark the cut surface of

the new growth with reddish points. Many of these blood-vessels, especially the smallest, possess a much-thickened wall (perithelium).

"This form of tumour is never sharply defined; on examining the edges by the microscope, one may see at points apparently normal a strongly marked cellular infiltration, which gradually passes into the healthy tissue.

"The yellow mass is found first in the form of veins, streaks, or so that one or several yellow dry nodules surrounded by the greyish-red substance project into the structure just described. This is

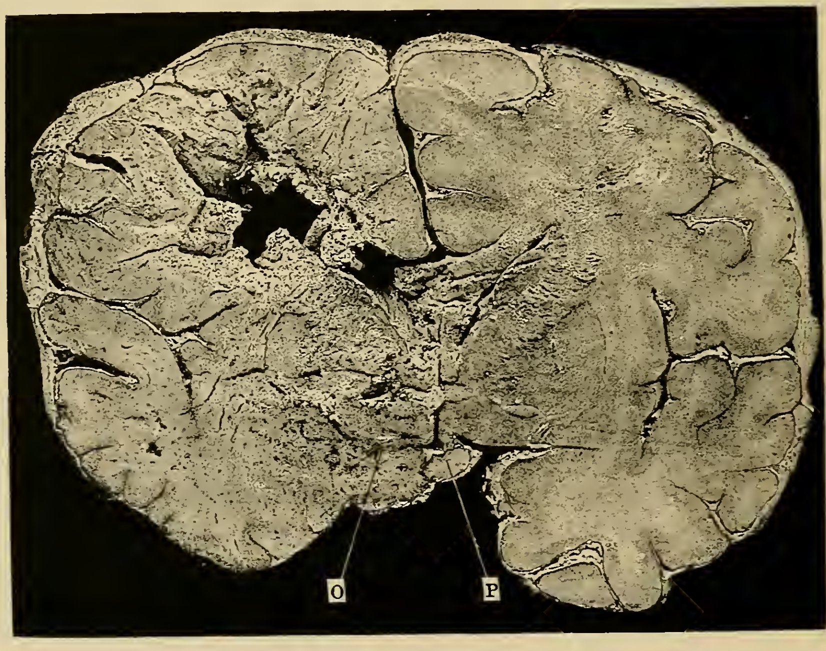

Fig. 82.—Transverse vertical section through mid-region of the brain in a case of cerebral syphilis, showing a gumma involving the right optic nerve and adjacent portions of the brain tissue, with extensive softening and disease of almost the whole transverse section of the right hemisphere.

The letter *O* points to the enormously swollen right, and *P* to the comparatively normal left optic nerve.

From a photograph (reduced in size).

particularly frequent where the new growth passes into nerve tissue, or is chiefly situated therein. It then consists either (1) of a dry, somewhat shining fibrous substance, appearing in thick sections, under the microscope, grey or greyish-yellow, the origin of which cannot with certainty be determined, but which may be in some cases

the remains of the original tissue compressed by the new growth (atrophic neuroglia), and, in others, masses of atrophic cells of the neoplasm itself; or (2)—especially in nerve sheaths, in the tissue of the dura mater, in the larger dry masses—of cells, round or spindle-shaped in a state of fatty degeneration, that is, masses of granule cells, which are situated in large agglomerations within the meshes of the original tissue.

"Secondly, we find them in the form of completely circumscribed, often almost encapsuled tumours (like cerebral tubercles), around

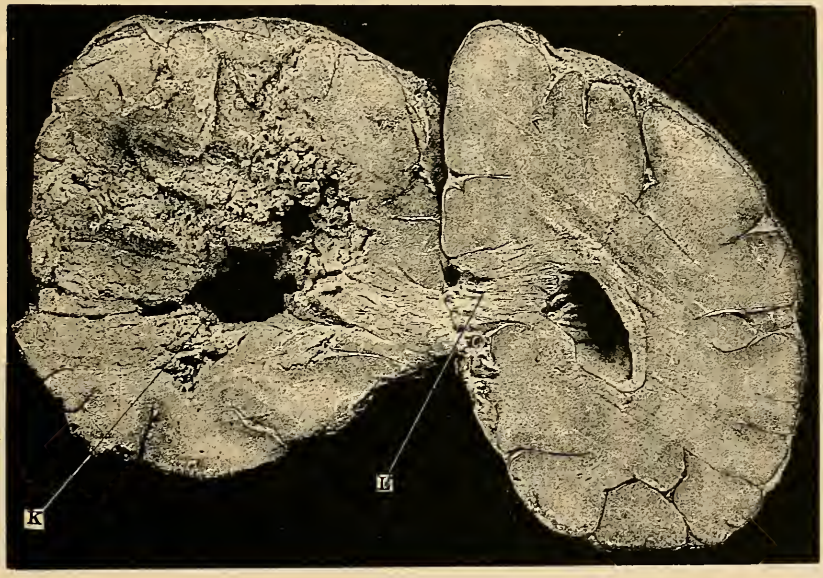

Fig. 83.—Transverse vertical section through the occipital lobes of the brain, in a case of cerebral syphilis, showing extensive softening, involving the whole of the transverse section of the right hemisphere.

The letter *K* points to a large softened cavity in the interior of the right hemisphere; the letter *L* to the corpus callosum on the left (healthy) side.

From a photograph (reduced in size).

which remains of chronic inflammation, or the grey-red new formation may be found. They are as large as an almond or dove's egg, and in the first case several often lie together. Their form is not infrequently determined by the space in which they are seated (fissures between cerebral convolutions), behaving thus simply like a caseous inflammatory exudation. Upon microscopic examination it seems that this homogeneous mass consists of a granular substance, entirely

uniform upon thin sections, apparently without further structure, and completely devoid of vessels. When broken up or torn apart, however, it sets free for the first time a multitude of angular or roundish granular elements, which resemble shrivelled or broken cells of roundish shape, and may correspond very well to the remains of the round cells which form the greyish-red syphiloma. Of the other

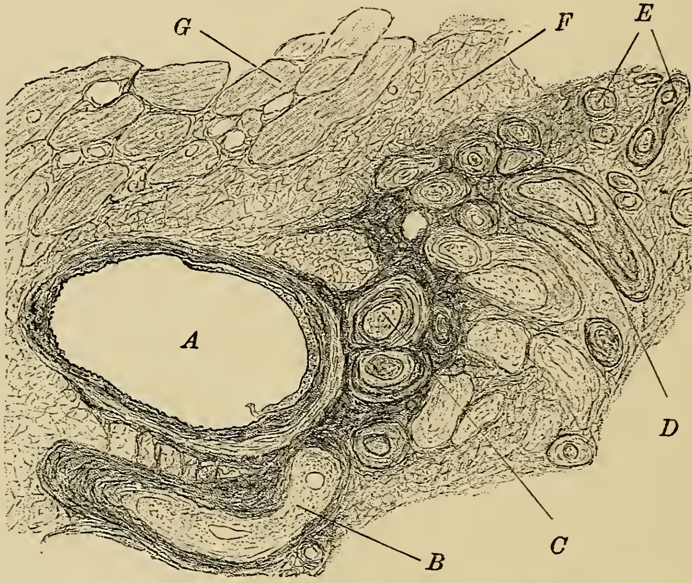

FIG. 84.—Camera lucida drawing of a microscopical section through a syphilitic gumma at the base of the brain, showing numerous vessels (*A*, *B*, *C*, *D*, *E*), the walls of which are greatly thickened, and many of which are entirely obliterated by syphilitic endarteritis. Hartnack, oc. 2, obj. 1 ; and drawing reduced from $4\frac{3}{4}$ to $3\frac{1}{2}$ inches.

G, bundles of the third nerve ; *A*, large artery transversely divided, its inner coat is slightly thickened ; *B*, small artery in longitudinal, and *C*, *D*, and *E*, small arteries in transverse section, showing extreme endarteritis obliterans. Most of these small vessels are completely obliterated by the thickening of their inner coat. The minute details of the lesion cannot of course be made out under this low power.

The letter *F* points to the inflammatory (gummatous) material in which the obliterated (and newly formed) vessels lie.

elements, connective-tissue fibres, spindle cells, &c., nothing more is to be seen. Here and there lie groups of needle-shaped crystals of fat, with granules and crystals of blood pigment intermixed, while nearer the edges of the yellow growth may be found upon section masses of fatty granulation cells. The chief part of the process, however, here does not consist in fatty degeneration, but in genuine cellular atrophy (caseification, tubercularisation, in the French sense). In the

interior such tumours are perfectly dry, or some small spots of softening may be found. The surrounding tissue always shows a recent, tolerably vascular, proliferating cell-mass, like that which we find in general in the greyish-red growth; and even where such a mass is encapsuled (which is always the case with the tumours situated in the dura mater), the meshes of the connective tissue forming the capsule are infiltrated with young cells.

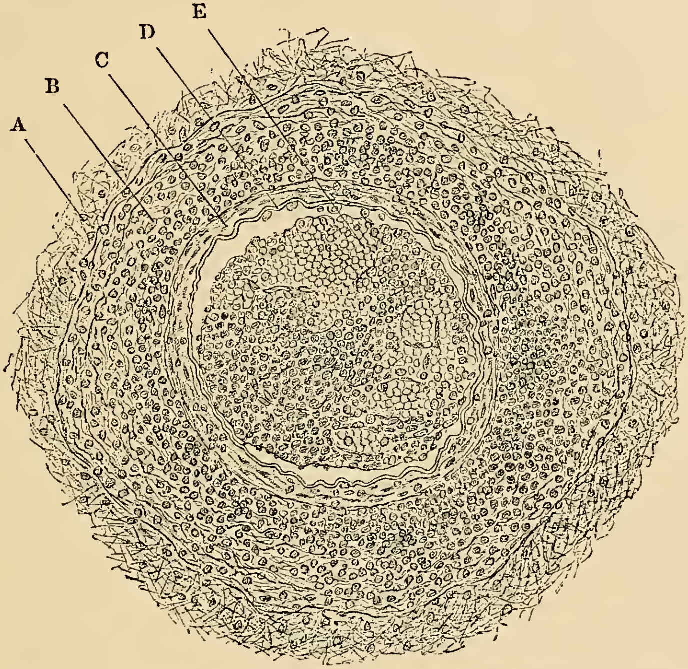

FIG. 85.—Camera lucida drawing of a microscopical section through a vessel in a case of cerebral syphilis, showing great infiltration and thickening of the outer coat (*B*), and an organising thrombus (*E*) in its interior. Hartnack, oc. 3, obj. 8, tube out; and drawing reduced from $4\frac{1}{8}$ to $3\frac{1}{4}$ inches.

A, inflammatory products round vessel; *B*, outer; *C*, middle; and *D*, inner coats; *E*, organised thrombus, containing delicate new-formed vessels in its interior.

"It is probable, if not yet strictly proved, that these yellow tumours originate in the greyish-red, and represent the dead and atrophied remains of the younger soft tissue.

"This neoplasm in its various forms has an entirely different signification, and even certain anatomical peculiarities, according to the locality within the cranial cavity in which it is developed.

"It has two favourite seats: the dura mater and the subarachnoid space.

"The growth very frequently affects the dura mater alone, without the participation of any other tissue in the cranial cavity. It then

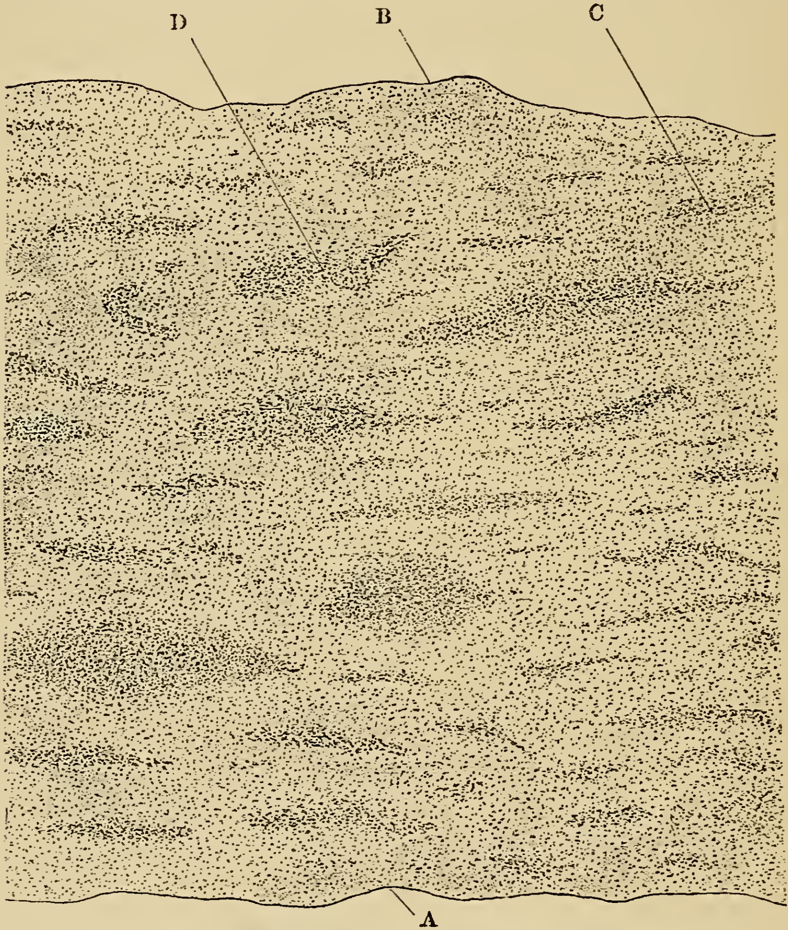

FIG. 86.—Camera lucida drawing of a microscopical (longitudinal) section through a bundle of the third nerve in cerebral syphilis, showing the nerve tissue entirely destroyed, and the nerve infiltrated with cellular elements. Hartnack, oc. 2, obj. 3; and drawing reduced from $4\frac{1}{2}$ to 4 inches.
A, *B*, free surfaces of nerve; *C*, *D*, masses of cells in nerve.

develops between the two (greatly thickened) layers of the dura mater, and is beautifully encapsuled thereby.

"The soft greyish-red growth is almost never met with in this place, but either the fibrous modification of the cellular neoplasm, or,

most frequently, the dry, yellow tumour, which may attain the size of a dove's or even a hen's egg. It may be developed in any region of the dura mater, but grows particularly large in the duplicatures, such as the falx, &c.

"One frequently meets with smaller, recent, but yet firm tumours in the dura mater overlying the sphenoid bone. In such cases the membrane is thickened around the tumour and for some distance in the neighbourhood, and the interstices of the connective tissue infiltrated with rows of round cells; the part of the cranial bones corresponding to the tumour is subject to the so-called caries sicca, is rough and eroded; the rest of the cerebral contents are normal."[1]

The naked-eye and microscopical characters of the gumma and other syphilitic lesions of the nervous tissues are well represented in Figs. 76–86.

Diagnostic characters of the syphilitic tumour.—The most important diagnostic indication of the syphilitic intracranial tumour is the presence of definite manifestations of syphilis in some other organ or tissue, such, for example, as intracranial or tibial nodes. Buzzard lays great stress upon the presence of a muddy complexion and other signs of a cachexia, not traceable to any visceral disease.

In those cases, in which there is no distinct evidence of associated syphilitic lesions, a clear history of syphilitic infection, and more especially of secondary symptoms, a cicatrix on the penis, or the remains of other syphilitic lesions, and the fact that there are no definite (positive) indications of any other form of new growth, are important.

In this connection, I must repeat that in many cases of cerebral syphilis the secondary symptoms are very slight, or sometimes altogether absent; certainly, in some cases, they pass unobserved by the patient. The fact that in women the primary sore may also pass unnoticed, or its significance may not be realised, and the circumstance that the denial of syphilis cannot always be relied upon, must also be remembered.

The facts that the patient is a male, between the ages of twenty-five and fifty; that the headache is worse at night; that there is

[1] Ziemssen's *Cyclopædia of Medicine*, vol. xii., p. 303.

tenderness on skull percussion; the presence of well-marked Jacksonian epilepsy (indicative of a cortical and irritative lesion); the presence of localised paralysis due to a lesion of an intracranial nerve (more especially paralysis of the muscles supplied by the third nerve); the occurrence of pseudo-apoplectic seizures or prolonged somnolent attacks; the fact that the arrangement of the symptoms is anomalous; the presence of many different symptoms which it is impossible to refer to a single cerebral lesion; and the marked relief which in many cases immediately follows vigorous anti-syphilitic treatment, are other points of great diagnostic importance. It is necessary to remember that the *secondary* effects of syphilitic brain lesions—such, for example, as softenings due to syphilitic disease of a large vessel—cannot be cured by anti-syphilitic remedies. On this point see page 224.

Gliomata.

After the scrofulous and syphilitic varieties, this is probably the most frequent form of brain tumour. It may occur in any part of the intracranial nerve centres (cerebrum, cerebellum, pons Varolii, or medulla oblongata), and usually, I believe, has its starting-point in the white rather than in the grey matter (see Fig. 87). Gliomatous tumours are very variable in size, but it is common to find large portions of the brain tissue infiltrated. Cases have come under my own observation in which considerably more than half of one cerebral hemisphere has been affected in this manner. This tendency to infiltrate the nervous structures is the most characteristic feature of the gliomatous tumour. The tumour tissue is never limited by a capsule, and it is impossible in many cases to say, without microscopical examination, where the tumour tissue ceases and the normal brain tissue begins.

The affected part of the brain often appears to be swollen, and is usually of a delicate pink, or purplish pink colour. In other cases the normal colour of the affected part is retained; in others again, nodules of firm, old, yellow and caseous-looking, or recent, black blood-clot are scattered throughout the affected tissue. The corpus callosum or basal ganglia may, in consequence of this gliomatous infiltration, be swollen to twice or three times their usual size.

FIG. 87.—Camera lucida drawing of a microscopical section through the cortex of the brain in a case of glioma. Stained with picro-carmine, cleared with oil of cloves, and mounted in xylol balsam ; magnified, Hartnack, oc. 2, obj. 1, tube in, and drawing reduced from $9\frac{3}{4}$ to $5\frac{3}{4}$ inches.

The gliomatous tissue is situated in the white matter, and has not involved the surface of the brain cortex.

The letter *A* points to the pia mater, which is healthy; *B*, to the superficial part of the convolution (grey matter), which is also healthy ; *C*, to a dilated blood-vessel in a portion of the grey matter adjacent to the gliomatous tissue; *D*, to a vessel passing into the grey matter from the surface of the brain ; *E*, to the superficial part of the tumour, which is invading the deep layers of the grey matter ; *F*, to vessels in the midst of the tumour—some of them are small, others large and varicose ; *G*, to a cavity in the centre of the section (probably artificial, and produced in preparing and mounting it) ; *H*, to the glio-

When large extravasations of blood have occurred just before death, the condition is easily mistaken for an ordinary hæmorrhagic apoplexy. A previous history of headache and vomiting, and above all, the presence of double optic neuritis—a condition which seldom, if ever, occurs in ordinary cerebral hæmorrhage—should lead the observer to suspect the true nature of the condition. Cysts, which are sometimes of large size, may be developed in connection with gliomatous tumours.

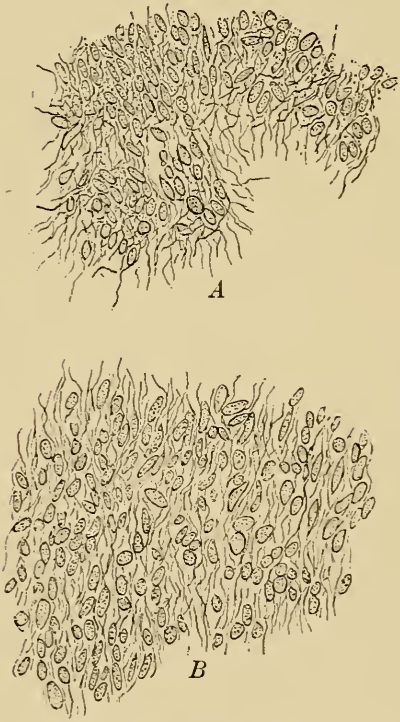

FIG. 88.—*A* and *B*, camera lucida drawings of portions of a microscopical section of a gliomatous tumour of the brain. Stained with picro-carmine, and mounted in Farrant's solution. Both the glial cells and glial fibres are well seen.

In some cases, the tissue of the tumour feels much firmer than in others, and two varieties of glioma—the hard and soft—have been described.

On microscopical examination, the tumour is found to be composed of small round or oval cells, and of extremely delicate fibres (glial threads); the exact appearance which the microscopical section presents, depends upon the method of preparation, the ramifying thread-like processes, which project in all directions from the cellular elements, being best displayed by the "half-clearing" method of preparation.[1]—(See Figs. 88 and 89.)

Blood corpuscles are often infiltrated around the tissues of the tumour. In several instances, I have found in the substance of the tumour large round cellular elements, some of which were distended with clear fluid contents, others of which contained red blood

[1] The reader who is interested in these histological details will find a full description of this method of preparing nerve tissues in the *Edinburgh Medical Journal* for October 1886, p. 322.

corpuscles in their interior (see Fig. 90). In many cases sarcomatous elements are to be found in some parts of the tumour, and in some

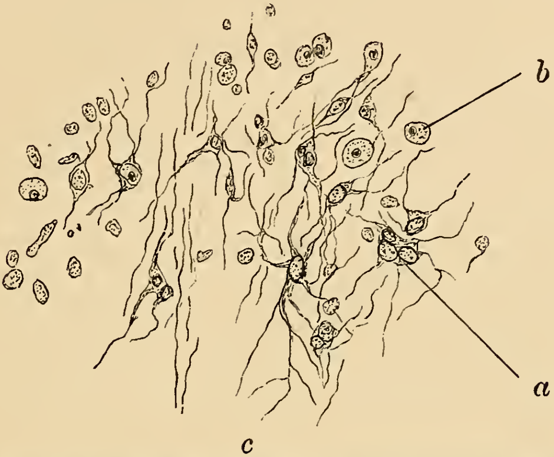

FIG. 89.—Section through a portion of a gliomatous tumour, showing a varicose and aneurismally dilated vessel surrounded with glial cells (half-cleared).

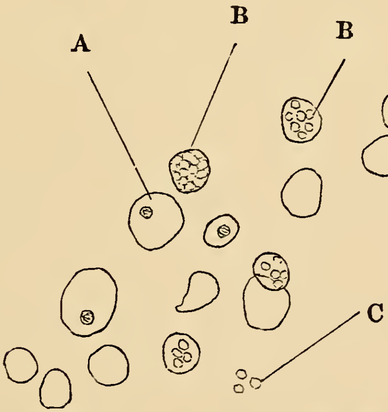

FIG. 90.—Camera lucida drawing of a microscopical section of a portion of a gliomatous tumour, showing large round transparent cells, some of which contain a single nucleus, others red blood globules. These cells lay in the midst of extravasated red blood corpuscles, which have been omitted from the drawing.

Stained with picro-carmine, half-cleared with methylated spirit and oil of cloves, and mounted in xylol balsam. (Magnified—Hartnack, oc. 3, obj. 8.)

A, large cell containing a single nucleus; *B*, *B*, cells containing red blood corpuscles; *C*, red blood globules.

they are so numerous that the tumour is rightly termed a glio-sarcoma or a sarco-glioma. Hyaline degeneration of the vessels and

myxomatous degeneration of the tissues of the tumour are not uncommon.—(See Figs. 91-94.)

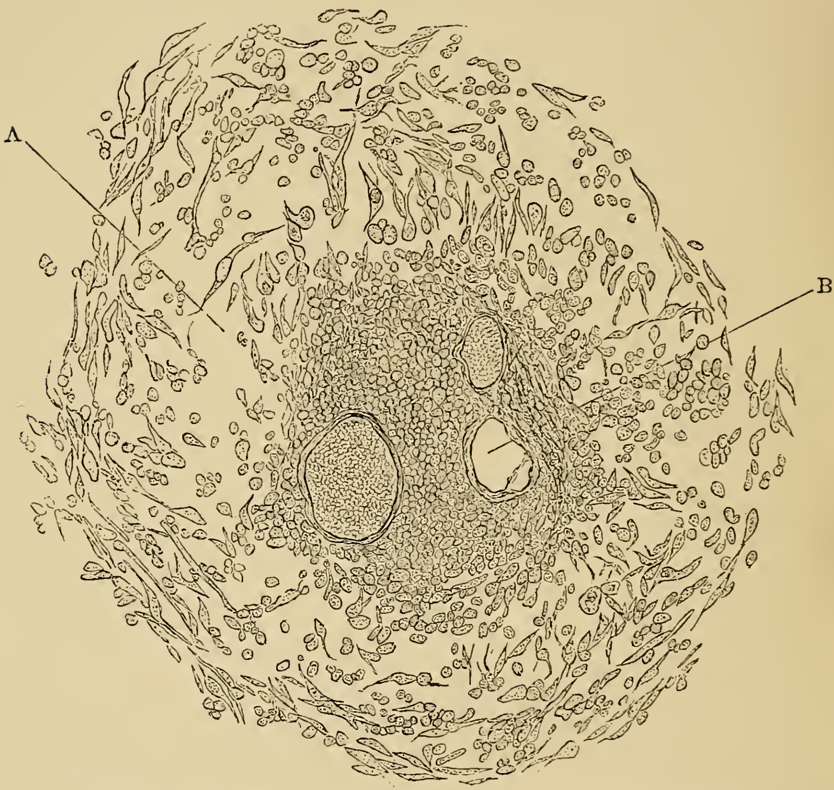

FIG. 91.—Camera lucida drawing of a portion of a section of a sarcomatous tumour of the brain, which has undergone myxomatous degeneration. Stained with picro-carmine, and mounted in Farrant's solution ; magnified, Hartnack, oc. 3, obj. 8, tube out, and drawing reduced from 6 to 4 inches.

In the centre of the figure three blood-vessels, one of which (*B*) is empty, are surrounded by a dense mass of small cells. The adjacent portion of the section is composed of large irregular and spindle-shaped cells embedded in a clear gelatinous fluid, which cannot, of course, be shown in the illustration. The letter *A* points to a space filled with this fluid, and in which no cells are seen.

One of the most important structural characters of gliomatous tumours, from a clinical point of view, is the enormous vascularity which they in many instances present. In places the tumour tissue may appear to be composed entirely of large thin-walled vessels, in the interstices between which a few glial cells are scattered.

Aneurismal and varicose dilatations of the vessels may often be noticed in such cases.—(See Figs. 41 and 42, pages 118 and 119.)

FIG. 92.—Camera lucida drawing of large branching and ribbon-shaped cells from a section of a sarcomatous tumour of the brain, which had undergone myxomatous degeneration. Stained with picro-carmine, mounted in Farrant's solution; and magnified, Hartnack, oc. 3, obj. 9 immersion, tube out, and drawing reduced from 4 to $3\frac{3}{8}$ inches.

The cells, some of which contain several nuclei, are embedded in a clear gelatinous fluid, in which the transversely divided fibres of cell processes are seen as fine dots.

In some cases of glioma, Osler found large cells and fibres, which he describes as follow :—" (1.) The ' spinnen' or spider cells (characteristic of glioma), which present variations in size: (2.) Large spindle-shaped cells, with single large nuclei (some of the largest cells met with in tumours): (3.) Cells like the ganglion cells of nerve centres, with large nuclei and one or more processes; they are larger than the spider cells: (4.) Translucent band-like fibres, tapering at each end, without nucleus or granular protoplasm, regarded as vitreous or hyaline transformation of the large spindle cells." I have in two or three cases met with cells and fibres resembling those described by

Osler in sarcomata or glio-sarcomata, which had undergone the myxomatous form of degeneration (see Figs. 91 and 92).

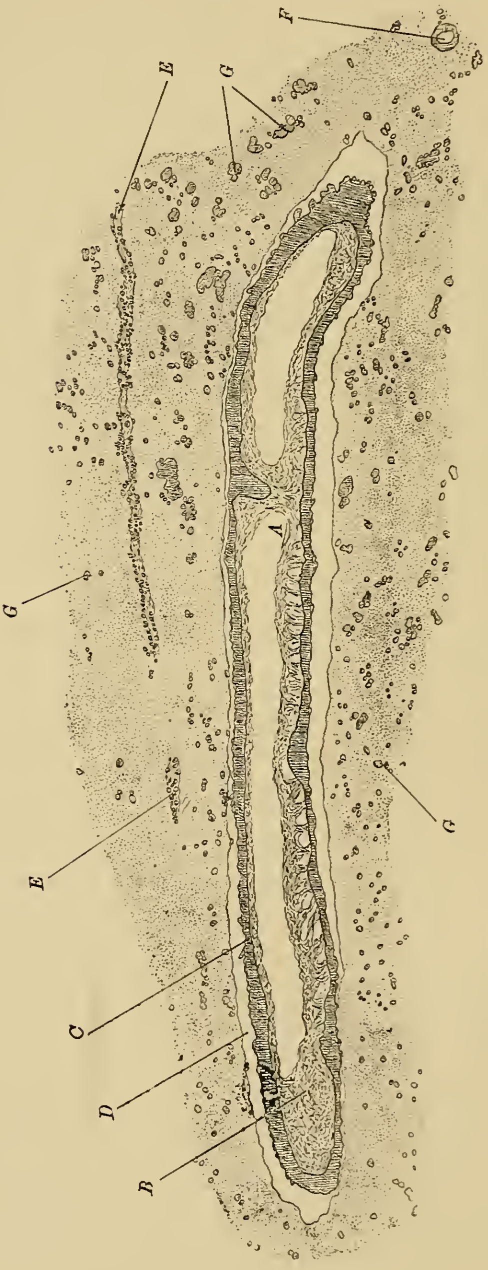

FIG. 93.—Glio-sarcomatous tumour with hyaloid degeneration of vessels. Longitudinal section through a vessel (*A*) of some size, the walls of which are infiltrated with hyaloid material, and the cavity of which is partly obliterated by a thrombus. The vessel also contained blood corpuscles, which are not represented in the drawing.

A, vessel; *B*, thrombus; *C*, hyaloid infiltration of coats; *D*, lymphatic space; *E*, *E*, small vessels, to the outer surface of which numerous small colloid lumps are attached; *F*, small vessel in transverse section; *G*, *G*, colloid lumps.

"Klebs (quoted by Osler) holds that the ganglion-like cells are derived from the nerve cells of the grey matter, and that in the development of this variety of glioma all elements of the nerve tissue participate. Osler examined the advancing region of the tumour, and was not able to satisfy himself that the nerve cells were in process of proliferation. He thinks the large cells are connective tissue elements."[1] With this opinion my own observations entirely agree.

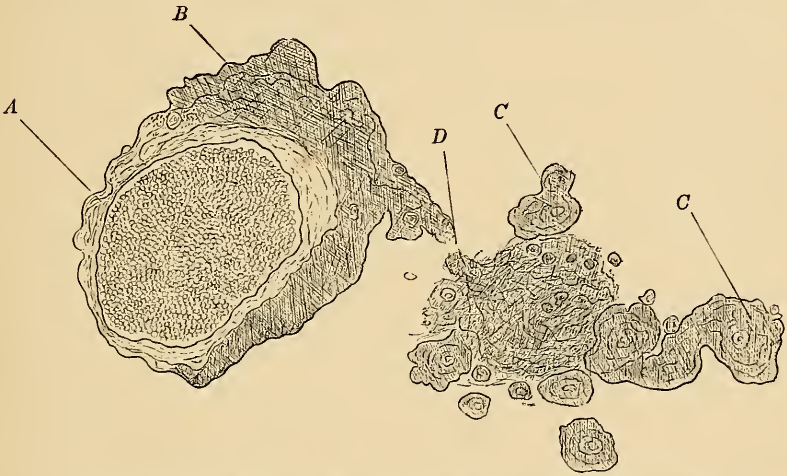

FIG. 94.—Glio-sarcomatous tumour ; hyaloid degeneration of vessels. The letter *A* points to a vessel of some size (filled with blood corpuscles), the walls of which are partly infiltrated with hyaloid material (*B*), which is continuous at *D* (broken in mounting), with hyaloid lumps in the surrounding tissue of the tumour, the structure of which has, for the sake of clearness, been omitted from the drawing. The letters *C C* point to small blood-vessels, the walls of which are enormously thickened and infiltrated with the hyaloid material.

For a more detailed account of the histological characters of this very interesting case, see the *Edinburgh Medical Journal*, February 1887, p. 689.

Numerous small scattered masses of myeline, which are evidently derived from broken-down nerve tubes, may in some instances be seen in the midst of (and more especially at the border of) the gliomatous tissue. In some cases the microscopical characters of the affected tissue so closely resemble those of chronic cerebritis, that the possibility of the tumour being due to some toxic irritant, possibly a micrococcus, has forcibly suggested itself to me.

In some instances, the tumour extends by direct contact to adjacent

[1] Pepper's *System of Medicine:* Article on "Tumours of the Brain, and its Envelopes," by Drs. Charles K. Mills and James Hendrie, p. 1047.

portions of the opposite cerebral hemisphere, with which it comes in contact; in other words, some gliomata are auto-inoculable (see Fig. 47, page 152).

The special diagnostic features of the gliomatous tumour.—Gliomata may occur at any age, but are, I think, more common in children and young adults than in old people. In some instances the exciting cause appears to be a head injury. The rapidity of growth varies very remarkably in different cases. I have seen a large portion of the brain tissue infiltrated, in cases in which the symptoms were of recent origin; in other cases which have come under my notice, the glioma had evidently been present for several years.[1]

In some cases, there is very great, and in others little or no increase of the intracranial pressure, and apparently very little cerebral irritation. The tumour may consequently in some cases be almost entirely latent; while, in others, the general symptoms (headache, vomiting, double optic neuritis, &c.) are very prominent.

Irritative phenomena, such as attacks of Jacksonian epilepsy, are not so common as in some other forms of tumours (*e.g.*, syphilomata); in consequence, I believe, of the fact that the new growth usually commences in the white substance, and destroys the nerve tubes before it produces irritation of the grey matter. Ziegler seems to differ from this opinion, for he states that gliomata are usually found close beneath the pia mater.[2]

A gradual and progressive paralysis without spasms is highly suggestive of a subcortical tumour, which is in many cases a glioma.

The general nutrition is, in many cases of glioma, well preserved. Pseudo-apoplectic attacks, and more especially repeated slight apoplectic seizures followed by paralysis (*i.e.*, apoplectic attacks evidently the result of hæmorrhagic extravasations, rather than of mere congestion), are very suggestive of this form of new growth.

In one instance which I have reported, imperfect hemiplegia, which varied in a remarkable manner from time to time, was evidently due to the pressure of a cyst (developed in connection with a glioma) upon the motor fibres of the pyramidal tract.[3]

[1] See cases reported in the *Edinburgh Medical Journal* for January 1887, pp. 591 and 623.

[2] *Special Pathological Anatomy*, section xii., p. 314.

[3] See cases reported in the *Edinburgh Medical Journal* for January 1887, p. 623.

Sarcomata.

All the different forms of sarcoma may occur within the skull. In some cases the new growth is primary, and has its starting-point in (*a*) the bones of the cranial wall, (*b*) the membranes, or (*c*) the connective tissue of the brain itself (in the form of the glio-sarcomatous or sarco-gliomatous tumour already described). In others the sarcoma is secondary to a similar form of new growth in some other part of the body, commonly in the lungs.

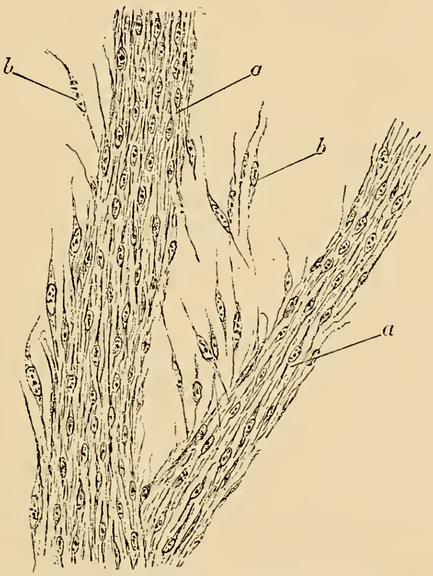

Fig. 95.—Camera lucida drawing of a microscopical section of a cerebral tumour, composed of extremely fine and delicate spindle cells. (Magnified —Hartnack, oc. 3, obj. 7, and tube out.)
The letters *a a* point to masses ; and *b b* to individual spindle cells.

The size of intracranial sarcomatous tumours is very variable, and depends to a large extent upon the primary or secondary nature of the condition. Primary tumours may attain to the size of the fist or even larger (see Figs. 1-4, pages 11 to 14). In some cases, the tumour is limited by a well-defined capsule; in others (sarco-gliomatous growths more especially), it infiltrates the nervous tissues. The effects produced upon the nervous structures are very different in different cases.

Slow growing, encapsuled sarcomata produce atrophy rather than irritation of the brain tissue with which they come into contact; in such cases, the general symptoms may be very severe, but the localising indications slight or altogether absent.

Rapidly growing sarcomata, which infiltrate the brain tissue, may produce marked irritative phenomena. In the remarkable case represented in Figs. 33 and 34, pages 109 and 110, the grey matter of the brain was so extensively involved, that "diffuse" cerebral symptoms were produced.

FIG. 96.—Cells from a myeloid sarcoma of the brain. (Magnified—Hartnack, oc. 3, obj. 8, tube out, and drawing reduced from 4 to 3⅝ inches.)

The microscopical characters vary very greatly in different cases, —see Figs. 33 to 38, pages 109 to 114, and Figs. 95 to 106, in which the structure of some of the more interesting varieties, which have come under my own notice, is shown.

Some forms of sarcoma (more especially sarco-gliomatous tumours) are remarkably vascular. Hæmorrhagic extravasations may occur in such cases, as in ordinary gliomata.

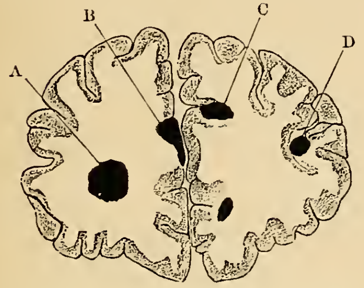

FIG. 97.—Transverse vertical section through the frontal lobes in a case of melanotic sarcoma, showing several deposits (*A*, *B*, *C*, *D*) of some size. (Reduced from a photograph.)

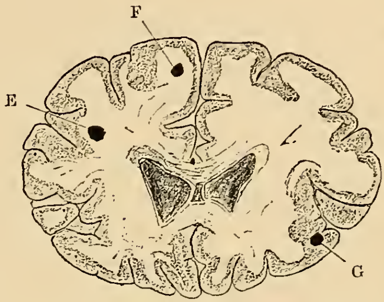

FIG. 98.—Transverse vertical section through the brain of S. E. M. at the level of the anterior end of the corpus callosum, showing three small melanotic masses (*E*, *F*, *G*). (Reduced from a photograph.)

The diagnostic features of sarcomata.—There are practically no special indications in the great majority of cases, unless the brain tumour be secondary to some well-marked sarcomatous lesion elsewhere.

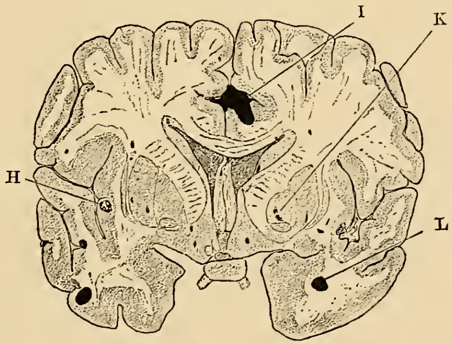

FIG. 99.—Transverse vertical section through the brain of S. E. M. at the level of the optic chiasma, showing numerous melanotic deposits, the majority of which are of small size. (Reduced from a photograph.)

The letter *H* points to a small nodule in the left island of Reil ; *I*, to a large superficial mass in the anterior longitudinal fissure ; *K*, to a small mass in the right lenticular nucleus ; *L*, to a nodule in the white matter of the right temporo-sphenoidal lobe. Many smaller nodules (which are unlettered) are seen in both hemispheres. In this section the nodules on the two sides of the brain are remarkably symmetrical in distribution.

Should symptoms, indicative of a tumour growing inwards from the bones or membranes, occur in a young person in whom there is

no suspicion of syphilis, and no likelihood (from the history or personal condition) of tubercle, the probability of the tumour being a sarcoma is considerable. The fact that the tumour had apparently

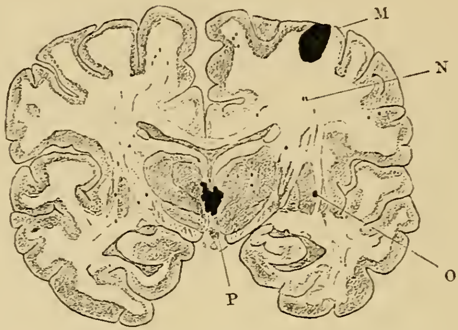

FIG. 100.—Transverse vertical section through the brain of S. E. M., just behind the optic chiasma, showing the position of the melanotic nodules. (Reduced from a photograph.)

The letter *M* points to a large nodule at the vertex ; *N*, to a small commencing nodule in the white matter of the centrum ovale ; *O*, to a small nodule in the right lenticular nucleus (there are several small nodules in the lenticular nucleus and external capsule of the left side) ; *P*, to a nodule of some size involving symmetrical parts of the optic thalami.

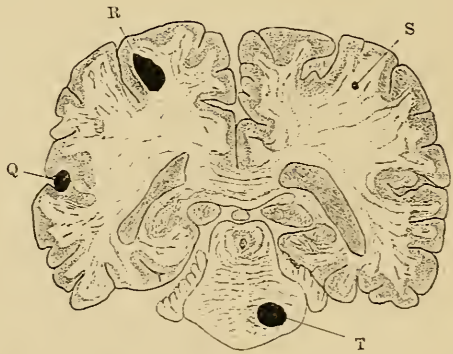

FIG. 101.—Transverse vertical section through the brain at the level of the greatest convexity of the pons Varolii, in a case of melanotic sarcoma, showing the position of melanotic nodules (*Q R S*) in the brain, and a large nodule (*T*) in the pons, involving the right pyramidal tract. (Reduced from a photograph.)

resulted from a head injury would, in my opinion, make the diagnosis of sarcoma still more probable. The general state of nutrition is, in many cases of intracranial sarcoma, well preserved.

Carcinomata.

Both primary and secondary cancers of the brain are comparatively rare. According to Obermeier primary cancer is the more common.[1]

Primary cerebral cancer usually originates either from the outer or inner surface of the dura. In the former case, the new growth may make its way through the bones of the skull and produce an external fungating tumour (such as is represented in Figs. 69, 70, and 71); in the latter, the cancer progresses inwards and invades the

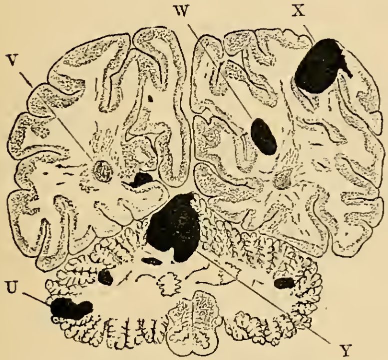

Fig. 102.—Transverse vertical section through the brain in a case of melanotic sarcoma, showing numerous melanotic deposits both in the brain and cerebellum. (Reduced from a photograph.)

The letter *U* points to a nodule of some size in the left lateral lobe of the cerebellum; *V*, to the left lateral ventricle; *W* and *X* to large nodules in the right cerebral hemisphere; *Y* to a large nodule in the middle lobe of the cerebellum.

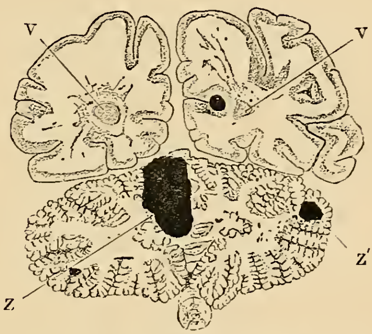

Fig. 103.—Transverse vertical section through the brain in the case of S. E. M., at the level of the lower end of the medulla oblongata, showing the position of the melanotic nodules. (Reduced from a photograph.)

The letters *V*, *V*, point to the lateral ventricles; *Z* to a large mass of new growth involving the middle lobe of the cerebellum; *Z'* to a small nodule in the right lateral lobe of the cerebellum.

brain tissues. It would appear that the dura is rarely, if ever, perforated, except at the seat of the openings which normally exist for the passage of blood-vessels or nerves.

Secondary cancers usually occur in the form of round nodules, several of which are often present in the same case (see Fig. 107, page 238, Fig. 7, page 32, and Fig. 56, page 171).

The microscopical characters of the new growth are the same as those of cancer elsewhere. In one instance I have met with the rare

[1] Ziemssen's *Cyclopœdia of Medicine*, vol. xii., p. 233.

form of new growth in which the cells were essentially epithelial in character, but the stroma made up entirely of delicate walled vessels. In the case to which I refer, and which is represented in Figs. 108, 109, 110, page 239 *et seq.*, the tumour had originated on the surface of the brain, apparently in the soft membranes; it had invaded and extensively infiltrated the brain tissue, a large portion of the left hemisphere being in the condition of "red softening." The term alveolar sarcoma is sometimes applied to this form of new growth.

FIG. 104.—Microscopical section through a portion of the white matter of the brain in the case of S. E. M., showing a nodule of melanotic sarcoma in an early stage. Low power (Hartnack, oc. 3, obj. 4, and tube out; drawing reduced from $3\frac{7}{8}$ to $3\frac{3}{8}$ inches).

The sarcomatous cells are massed round the blood-vessels; some of the vessels in the surrounding portions of the brain substance are very much dilated.

The letter *E* points to a mass of pigmented sarcomatous cells.

Ziegler describes its characters as follows:—"There are sarcomata which have a structure resembling that of gland tissue, or of epithelial new growths. This appearance of structure is due in part to the epithelial look of the cells, but chiefly to their aggregated arrangement in groups separated by fibrous septa. Such tumours are described as *alveolar sarcomata*. . . . Tumours of this kind occur chiefly in the skin, but they are also met with in the bones, lymphatic glands, and pia mater. . . . The way in which the alveolar structure is developed can often be clearly made out, especially in

tumours of the central nervous system. The normal intervascular tissue is transformed into masses of sarcoma cells, while septa are formed between the cell masses by the fibrous tissues lying along the course of the vessels. In other cases it looks as if a plexus of pre-existing or new formed vessels took on, as it were, an investment of

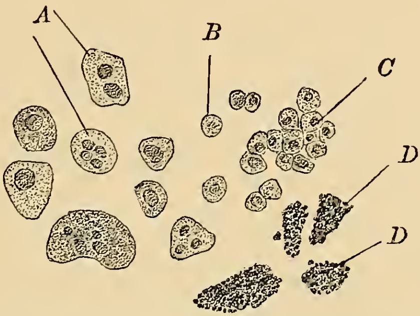

FIG. 105.—Individual cells from sarcomatous nodules in the case of S. E. M. (Magnified—Hartnack, oc. 3, obj. 8, and tube drawn out.)

The letter *A* points to large cells from a nodule in the dura mater ; *B* and *C*, to small round cells from a commencing nodule in the brain ; *D*, *D*, to large cells filled with large melanotic granules from a nodule in the tonsil.

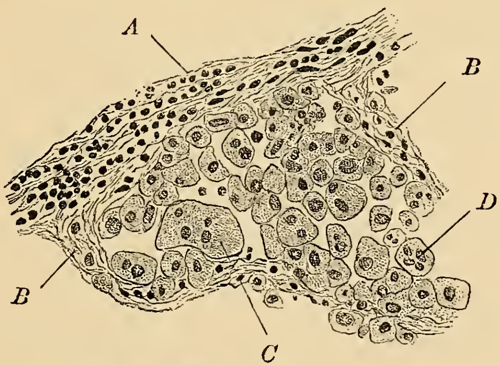

FIG. 106.—Microscopical section through a nodule of melanotic sarcoma attached to the dura mater in the case of S. E. M. (Magnified—Hartnack, oc. 3, obj. 8, and tube drawn out.)

The letter *A* points to the deepest layer of the dura (a small portion of which is only represented, for the membrane was very much thickened). The dura is seen to be infiltrated with deeply stained leucocytes and enlarged connective tissue corpuscles. The letters *B B* point to septa of fibrous tissue (which are also infiltrated with leucocytes) passing from the dura into the tumour tissue; *C* and *D*, to large sarcomatous cells containing several nuclei.

cells, and this grew thicker and thicker, till at length the inter-vascular spaces were entirely filled up. Accordingly we find this

form of new growth described as plexiform angio-sarcoma. It has also been described, and not infrequently, as *endothelioma.* On this view the cell nests arise by proliferation from endothelial cells. This certainly happens when masses of cells are formed from the

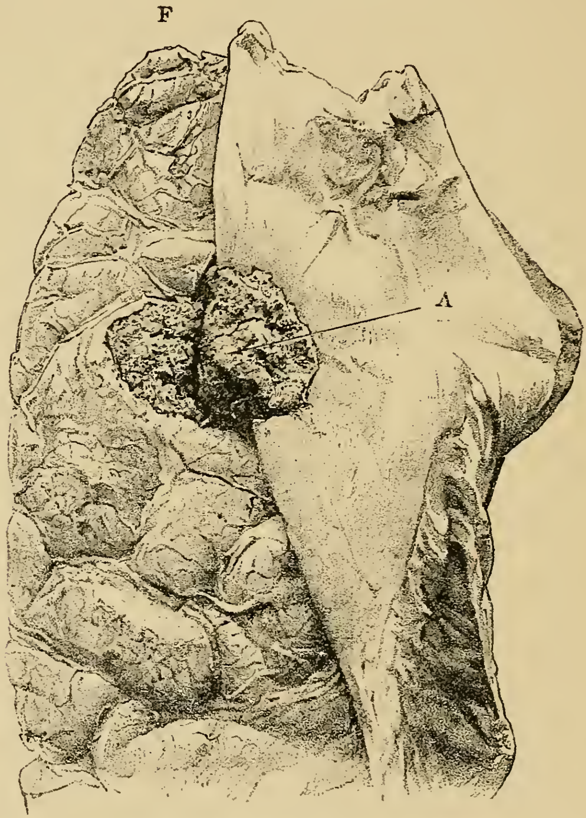

Fig. 107.—Surface of the frontal end of the left hemisphere of the brain in a case of disseminated cancer, showing a nodule of new growth (*A*) adherent on the one side to the dura, and on the other to the substance of the brain. (Copied from a photograph, and somewhat less than the actual size of the preparation.)

The letter *F* is placed at the anterior end of the frontal lobe; the letter *A*, which is placed on the reflected dura mater, points to the tumour.

endothelial covering of the subarachnoid mesh-work of the pia mater; the masses afterwards group themselves into "nests." Sometimes the proliferous endothelial cells of the pia mater are aggregated into small spherical nodules of a peculiar lustrous appearance. The tumour into which the membrane is transformed then contains small, shining, pearly bodies, made up of laminated layers of squamous or

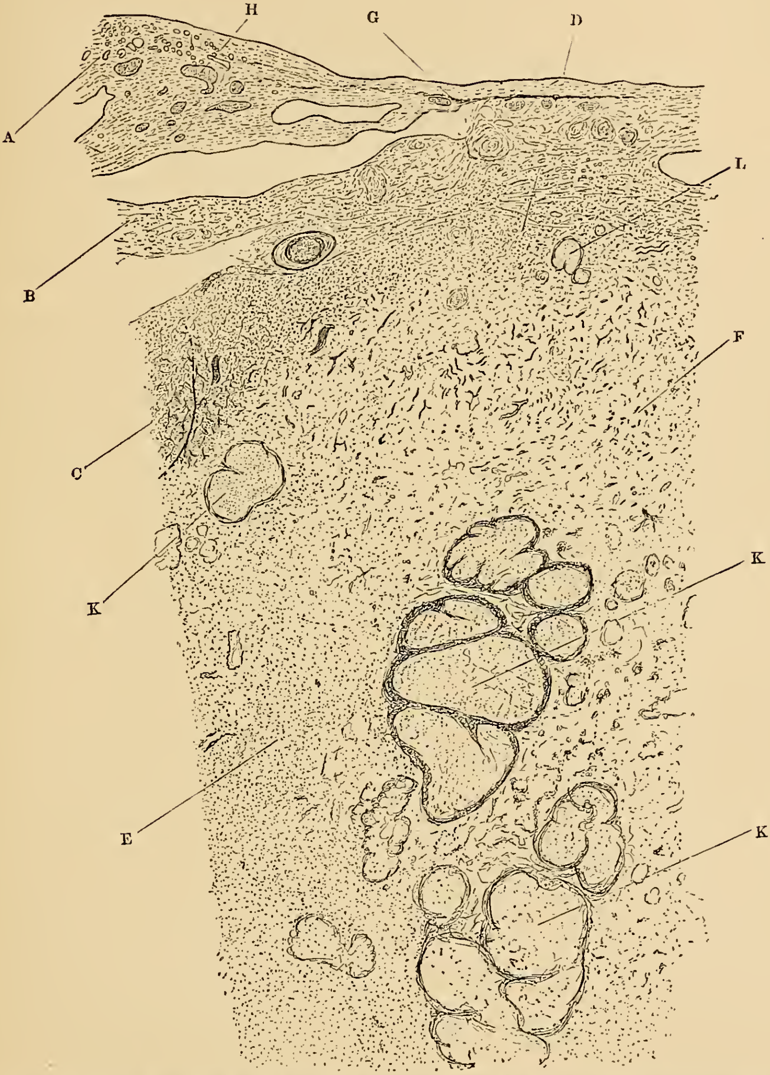

FIG. 108.—Camera lucida drawing of a section through a portion of the brain and its membranes in a case of plexiform angio-sarcoma (? vascular cancer). Stained with picro-carmine, cleared with oil of cloves, and mounted in xylol balsam. (Magnified—Hartnack, oc. 2, obj. 1, tube in, and drawing reduced from 12 to $5\frac{1}{2}$ inches.)

The letter *A* points to the greatly thickened dura mater, in which numerous vessels, some of large size (*H*), are seen; *B*, to the thickened soft membranes in which the tumour was embedded, and from which it seemed to have originated; *C*, to a portion of the tumour which is situated in the brain tissue; *D*, to the portion of the section which is more highly magnified in Fig. 110; *E*, to the deeper portion of the cortex which is in the process of being invaded by the new growth, and is in a condition of red softening; *G*, to a portion of the dura (not much thickened) which is shown more highly magnified in Fig. 110; *F*, to newly formed vessels in the superficial part of the cortex which is invaded by the tumour; *K*, *K*, *K*, to large hæmorrhagic extravasations in the cortex; *L*, to the hæmorrhagic extravasation, which is represented more highly magnified in Fig. 110.

tubular cells. Such tumours have been called *cholesteatomata* or pearly tumours." [1]

In Figs. 111 and 112, page 242, I have represented a remarkably fine example of an endothelioma, for which I am indebted to my friend Dr. T. W. McDowall, who has recorded the case in the *Journal of Mental Science*, April 1884.

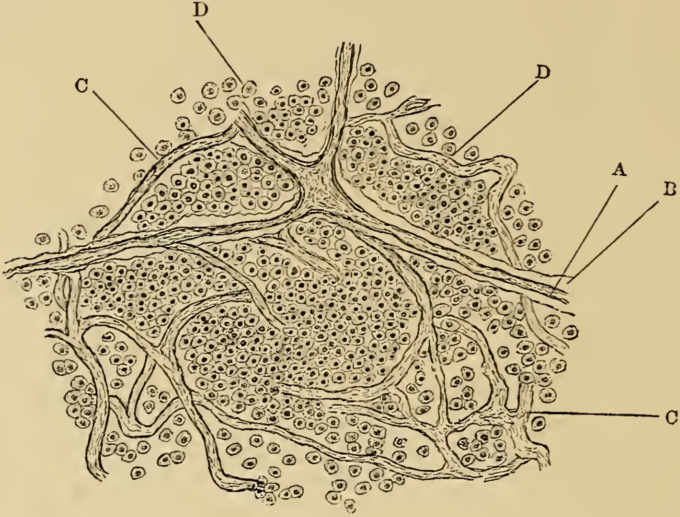

FIG. 109.—Camera lucida drawing of a microscopical section of a plexiform angio-sarcoma (? vascular cancer) of the brain. Stained with picro-carmine, cleared with oil of cloves, and mounted in xylol balsam. (Magnified —Hartnack. oc. 3, obj. 8, tube out, and drawing reduced from 5¼ to 3 inches.)
The tumour is composed of a network of thin-walled vessels, in the interstices of which round epithelial-like cells, each containing a single large nucleus, are densely packed.

The structure of this tumour seems to me essentially different from the vascular cancer or alveolar sarcoma represented in Figs. 108, 109, and 110.

The diagnostic indications suggestive of the cancerous nature of an intracranial tumour are in many cases insufficient to enable an opinion of any value to be arrived at. Perforation of the bones of the skull and the presence of an external fungating growth is, so far as I know, a certain sign of cancer or sarcoma. The fact that a cancerous tumour has been previously removed, or the presence of cancer in other organs, is likewise all-important. Failing these, the facts that the patient is old, and that he inherits a strong tendency to cancer, are

[1] Ziegler's *General Pathology*, p. 220.

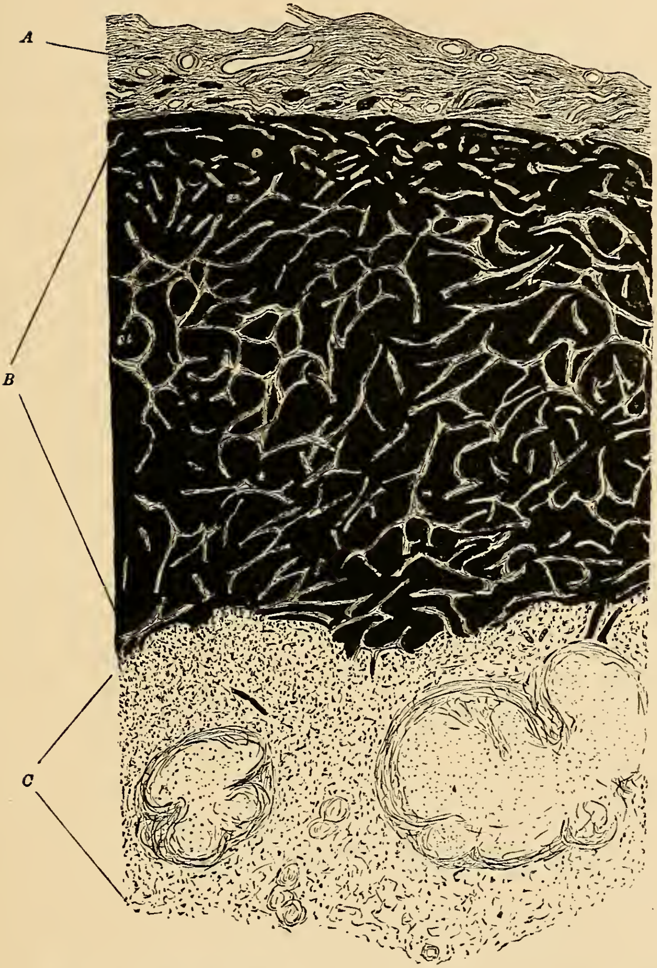

FIG. 110.—Camera lucida drawing of a microscopical section through the membranes and cortex of the brain in a case of plexiform angio-sarcoma (? vascular cancer). Stained with picro-carmine, cleared with oil of cloves, and mounted in xylol balsam. (Magnified—Hartnack, oc. 3, obj. 4, tube out, and drawing reduced from 10 to 5 inches.)

The letter *A* points to the thickened dura mater ; *B*, to the new growth, which is situated between the dura and the surface of the brain (*C*).

The tumour is composed of a network of thin-walled vessels well shown in the figure, the interstices of which are filled with cells not represented in the figure (in which the solid tissue of the tumour has been shaded black so as to bring out the vessels).

The brain tissue was extensively invaded by the tumour, and there were numerous hæmorrhagic extravasations (two of which are represented in the drawing) in the brain cortex. The greater part of the left hemisphere of the brain (in which the tumour was situated) was in a condition of red softening.

The cervical glands were much enlarged and infiltrated with the same form of new growth.

suggestive (though by no means very strong evidence) that the intra-cranial tumour is cancerous. The negative evidence, that there are no positive indications of any other form of new growth is also of some value.

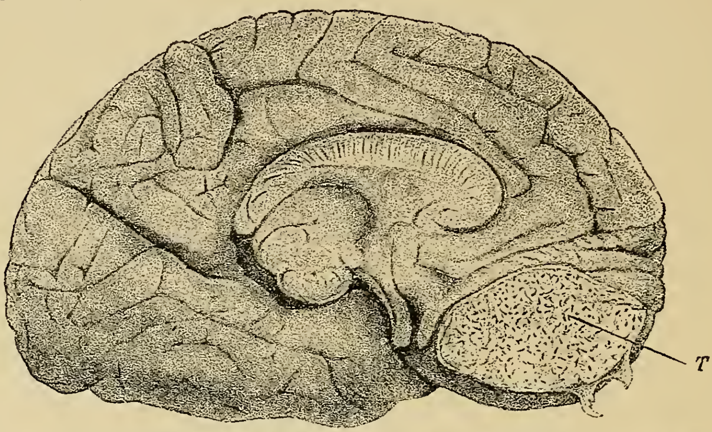

Fig. 111.—The left hemisphere of the brain in Dr. T. W. McDowall's case of endothelioma.

The letter *T* points to the tumour, which is situated at the under surface of the tip of the frontal lobe.

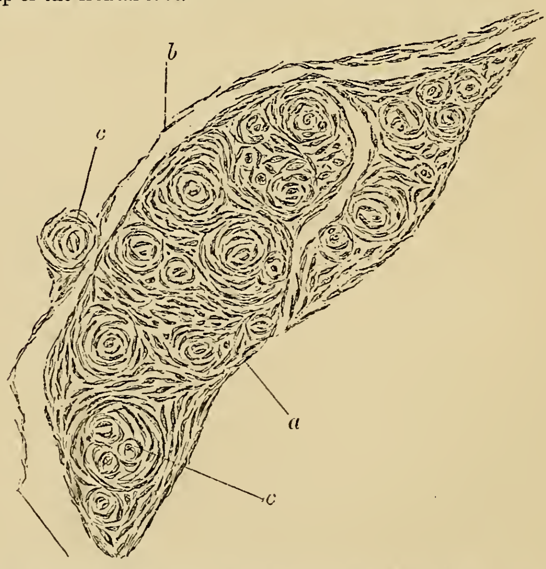

Fig. 112.—Camera lucida drawing of microscopical section of the endothelioma represented in Fig. 111. Stained with picro-carmine, cleared with oil of cloves, and mounted in xylol balsam. (Magnified—Hartnack, oc. 3, obj. 8, and tube out.)

The letter *a* points to the endothelial cells, seen as small spindles between the cells' nests, *c c;* the letter *b* to the edge of a large mass of the tumour, which is not shown in the figure.

Rare and unimportant forms of Tumour.

Hyperplasia of the pineal gland occasionally occurs. No case has come under my own observation. According to Obermeier, it is formed upon essentially the same structural type as glioma; the tumour may be as large as a walnut, or even larger.[1]

The symptoms may be partly due to pressure upon the corpora quadrigemina, and partly to compression of the veins of Galen and resulting hydrocephalus.

Psammomata, which occur in the form of small, hard, often stony tumours, usually about the size of a cherry stone, are of little or no clinical interest, for they very rarely give rise to symptoms. They spring from the membranes of the brain, are usually situated at the base, and on microscopical examination are found to contain sand-like particles identical with those which are normally found in the pineal gland.

Simple *fibrous*, *osseous*, and *fatty* tumours are occasionally, but very rarely, met with within the cranial cavity.

Melanoma is a rare pigmented tumour, which, according to Virchow, has its origin in the pigment cells of the dura mater. It is usually of small size, is sometimes multiple, but does not seem to be attended with any symptoms.

Neuroma is the term given to a genuine hyperplasia of the grey substance, which, according to Obermeier's description, "may be found in the shape of small tumours, varying in size from that of a millet-seed to that of a pea, and situated on the ventricular surface, in the white substance, or on the outer surface of the brain." No tumour of this description has come under my notice. The condition has been very rarely observed, and only in persons with some mental aberration, congenital or acquired. As Obermeier suggests, it may well be doubted whether the neuromatous tumours were in such cases the cause of the mental condition. Be this, however, as it may, when viewed as a cerebral tumour, the neuroma is a condition of no clinical importance.

The different forms of cyst have been previously alluded to, and need not be described.

[1] Ziemssen's *Cyclopædia*, vol. xii., p. 234.

CHAPTER X.

PROGNOSIS—DURATION, COURSE, AND TERMINATION—TREATMENT.

The prognosis in all cases of intracranial tumour is most uncertain, inasmuch as there is always a liability to sudden death. But barring this accident, which cannot be foreseen, it may be stated as a general rule that the progress of the great majority of cases is steadily downwards, with perhaps periods of remission or improvement. The prognosis is therefore as a rule most unfavourable. In confirmation of this statement, I may add that the remains of old cured tumours—other than the remains of former syphilitic lesions—are very rarely indeed found post mortem. I have myself only come across one case of this kind in at least a thousand autopsies. A microscopical section of this specimen—a small, hard tumour in the cerebellum, about the size of a threepenny piece—is represented in Fig. 113.

In syphilitic cases, remarkable improvement often results from a vigorous anti-syphilitic plan of treatment, and in some cases a complete cure seems to be effected; and in tubercular cases arrested development of the new growth, but very rarely complete cure, occasionally occurs. But even in the most favourable cases, the syphilitic lesion is liable (as the result, perhaps, of an injury to the head, or other exciting cause) to take on renewed activity, and a relapse to occur.

It is important, too, to realise and emphasise the fact that in cases of syphilitic nerve lesion the symptoms for the most part depend upon the *secondary* alterations (softenings, destructions, &c.) which the lesion produces in the nervous structures; and that, although anti-syphilitic treatment may be able to remove the primary (syphilitic) lesion, it is quite powerless to restore or renew the nerve tissue which

has been damaged or destroyed by that lesion. Hence in syphilitic cases, although the prognosis as regards life should never perhaps be hopeless, however desperate the condition may appear, the opinion as regards complete recovery, removal of paralysis, &c., depends upon

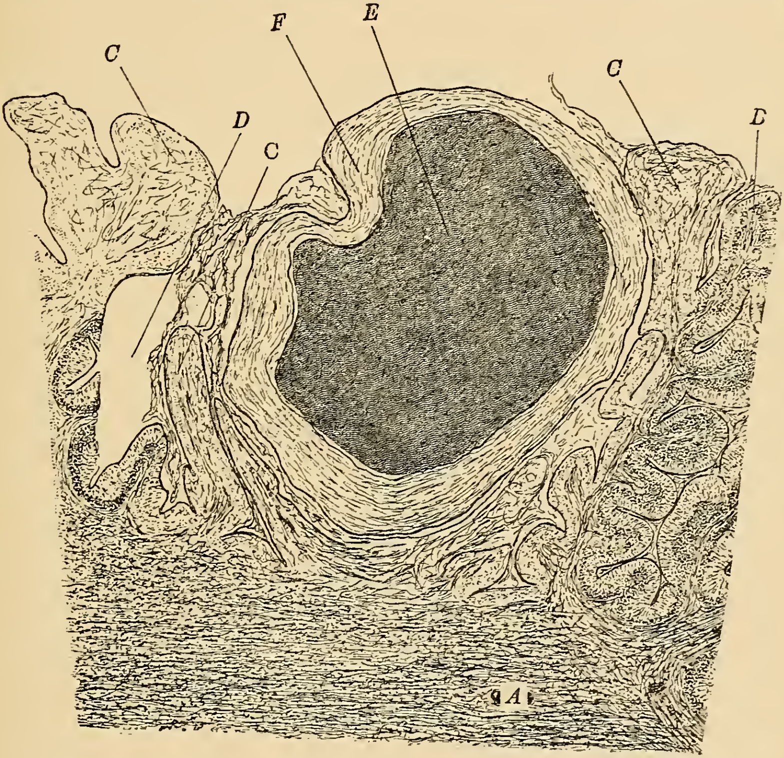

FIG. 113.—Camera lucida drawing of a microscopical section through a portion of the cerebellum, showing an old cured tumour, surrounded by a dense mass of fibrous tissue. (Magnified—Hartnack, oc. 3, obj. 1; tube in; drawing reduced from $5\frac{3}{4}$ to 4 inches.)

The letter *E* points to the tumour, which contained the remains of large epithelial-like cells, and which was densely infiltrated with calcareous particles; *F*, to the thick fibrous tissue surrounding the tumour; *A*, to the white matter of the cerebellum; *B*, to the normal grey matter of the cerebellum; *C C*, to atrophied portions of the cerebellar cortex; *D*, to a cavity, which is partly artificial (due to a portion of the normal cerebellar tissue having fallen out of it during the act of preparation), but which was partly filled with dense fibrous tissue.

In the atrophied portions of the cerebellum (*C C*) the normal nerve tissue has been entirely destroyed.

the amount and kind of damage to the nervous structure proper, as well as upon the period of the disease at which the treatment is

commenced. In a case of hemiplegia, due to extensive softening of the centrum ovale, the result of syphilitic disease of the cerebral vessels, the prognosis *as regards the removal of the paralysis* is little, if at all, better than in a case of hemiplegia due to hæmorrhagic extravasation or embolic blocking. For even if the syphilitic arterial disease could be removed by anti-syphilitic remedies, the nervous elements which have been softened and destroyed cannot be restored to their former condition. Again, it must be remembered that the cicatrix, which is left behind a cured syphilitic gumma, may be the source of a long-continued or permanent epilepsy.

When, however, the alteration in the nervous elements falls short of complete destruction—when, for instance, there is simply impaired nutrition as the result of deficient (and not completely arrested) blood supply—most marked improvement may result from anti-syphilitic remedies, the removal of the endarteritis or the periarteritis allowing of a freer blood supply to the nervous elements. Syphilitic disease of the larger vessels is said to resist anti-syphilitic remedies. Be that as it may, I am quite satisfied that the syphilitic lesions of the smaller vessels may be removed by anti-syphilitic treatment.

Again, in those cases in which the symptoms depend upon irritation rather than upon destruction of the nervous tissue (and in which enlargement of the connective tissue corpuscles, and infiltration of the nervous structures, in the immediate neighbourhood of the gumma, with nuclear elements, are the most striking histological changes), the most striking and brilliant results may often be obtained by a vigorous anti-syphilitic treatment.

In those cases, too, in which the peripheral nerve trunks (such as the trunk of the third nerve) are extensively affected by syphilitic infiltration, most remarkable restoration—removal of paralysis, and recovery of motor power—is in many cases effected.

In watching the effects of treatment in syphilitic as well as in other forms of tumour, it is important to remember that remarkable remissions and periods of apparent recovery not infrequently occur. In the remarkable case of sarcoma shown in Figs. 2, 3, 4, pages 12, 13, and 14, the improvement under treatment was most striking; the patient expressed herself as feeling quite well, and had indeed made arrangements to leave the hospital the very morning following the sudden epileptiform seizure which proved fatal.

In addition to the pathological character of the new growth, the possibility of its being removed by surgical interference is perhaps the most important element in forming the prognosis, but this point will be more appropriately considered under the head of treatment.

The causes of death are various. In some cases, the patient dies suddenly in an epileptic or apoplectic fit, or in one of the pseudo-apoplectic attacks which do not depend upon hæmorrhagic extravasation. Tumours in the neighbourhood of the medulla oblongata may probably cause sudden death by suddenly arresting the respiration or the heart.

Dr. Hughlings Jackson states that sudden deaths may be due to the violence of the pain and suffering. He states, "this has happened in a case of cerebellar abscess from ear disease,—the abscess had not ruptured." And again, "intense persisting pain, in cases of optic neuritis, makes one fear that the patient may die suddenly." [1] That is what we might, *a priori*, expect, for there is every reason to suppose that sudden arrest of the action of the heart may be produced by intense pain, fear, &c.; and in some cases of intracranial tumour the headache is most intense, and occurs in sudden paroxysms.

In other cases of intracranial tumour, death is gradual, either as the result of exhaustion, long-continued suffering, the pressure of the tumour upon important parts, such as the pons Varolii or medulla oblongata, the gradual deterioration of the mental faculties and bodily functions which accompanies copious ventricular dropsy, or in consequence of the presence of associated lesions in the great viscera of the thorax or abdomen (tubercle in the lungs or intestine, cancer in the stomach or liver, for example). Respiratory complications (bronchitis, œdema of the lungs, &c.), bedsores, or cystitis are in many cases the immediate cause of death.

Treatment.

The drug treatment of intracranial tumours is, speaking generally, most unsatisfactory. Syphilitic tumours may, in most cases, be materially benefited, and in some completely cured, by anti-syphilitic remedies—iodide of potassium and mercurials. The iodide must be

[1] *Transactions of the Ophthalmological Society of the United Kingdom*, vol. i., p. 75.

given in large doses, at least thirty grains three times daily. I have again and again seen most striking results obtained by these large doses, when smaller quantities (five or ten grains) have been previously given with little or no benefit. The administration of five-grain doses of iodide of potassium in cerebral syphilis is, in most instances, useless. If thirty grains three times daily is not followed by marked improvement, I am in the habit of increasing the dose to thirty grains four, five, or six times a day; and if the iodide alone does not relieve, I prescribe, in addition, mercurials (either in the form of corrosive sublimate internally, or as mercurial inunction externally). I agree with Buzzard in thinking that, in some cases in which there is no improvement under the iodide alone, the addition of mercurials is followed by marked benefit.

In syphilitic cases I am in the habit of continuing the iodide for weeks or months; and I have never seen any injurious effects from large doses given in this way. In one of my cases, the patient took thirty grains three times daily for at least twenty months without the slightest appearance of any bad effect.

Wood speaks highly of sarsaparilla, given in the form of a mixture of equal parts of the compound fluid extract and the compound syrup of sarsaparilla of the American Pharmacopœia. He states that the compound syrup of sarsaparilla covers the disagreeable taste of the iodide of potassium better than any other vehicle with which he is acquainted.[1]

In some tubercular cases, the iodide of potassium seems beneficial; in such cases I usually give it in smaller doses than in cases of syphilis (five-grain doses for an adult three times daily), together with cod liver oil, and other general remedies which are useful in phthisis.

Possibly the inunction of iodoform ointment into the scalp, which is said to have been followed by a cure in some cases of tubercular meningitis, or the internal administration of iodoform in the form of pills, may prove beneficial in some tubercular brain tumours.

In some cases of sarcoma, arsenic seems to restrain the development of the new growth. I cannot, however, say that I have ever seen any distinct relief from the administration of this remedy in cases of intracranial sarcoma.

[1] Pepper's *System of Medicine*, vol. v., p. 1017.

To sum up, the drug treatment of intracranial tumours is most unsatisfactory, except in the case of syphilitic growths. Iodide of potassium is the only remedy, so far as we at present know, which is likely to effect much benefit, and it should be fairly and freely tried in all cases, whether syphilitic or not, in which drug treatment is indicated or is likely to hold out any prospect of success.

Operative treatment.—The brilliant results which Macewen has obtained in cerebral surgery, and which Victor Horsley has recently published, seem to prove that the expectations which Hughes Bennett formed when he first advocated the operation were well founded. And, thanks to antisepsis, the dangers of trephining are now so slight, that in all cases of intracranial tumour in which substantial improvement is not obtained by the administration of iodide of potassium, the possibility of removing the new growth by operative procedure must be carefully considered.

The following appear to me to be the most important conditions for successful operative interference :—

(1.) *Satisfactory localisation of the tumour.*—It is quite obvious that, in the absence of localising symptoms, it may be impossible to come to any conclusion as to the position of the new growth. Under such circumstances, if the symptoms are very severe, and if they cannot be relieved in any other way, it may perhaps in some cases be advisable to trephine, as a palliative measure (*i.e.*, with the object of giving temporary relief); but, in the absence of any definite knowledge of the position of the new growth, an operation for its removal is of course contra-indicated.

(2.) *The tumour must be accessible.*—It is quite obvious that the risks attending attempts at removal of tumours in some situations, such as the pons Varolii, medulla oblongata, middle lobe of the cerebellum, and base of the brain, render operative procedure quite impracticable.

Tumours in the motor area, both because of the exactness with which they can in many cases be localised, and the facility with which they can be reached, offer the most favourable chance of removal by operation.

(3.) *The tumour must be single, and of such a pathological character as to permit of complete enucleation or removal.*

In the case of multiple tumours, such as secondary deposits of cancer or melanotic sarcoma, operative measures for the removal of the tumour are of course quite out of the question. Hence the great importance of exact diagnosis, not only as regards the position of the tumour, but also as regards the pathological character of the new growth.

It will be apparent from the foregoing statement, that in some cases the position of the tumour cannot be satisfactorily determined, that in others it is so situated that it cannot be safely and satisfactorily operated upon, and that in others it is of such a pathological character that it cannot be completely removed by means of operation. Hence the proportion of cases, in which the conditions for successful removal and cure by means of operation are present, is probably not great.

On the other hand, it must be remembered that in the great majority of cases of intracranial tumour, drugs are powerless to effect a cure, and that the inevitable termination, if left alone, is (after a longer or a shorter period, which is often attended with great suffering) death.

Further, that the determination of the *exact* nature and extent of the tumour by means of the symptoms is in many cases impossible.

Hence, in many cases, the surgeon is justified, if iodide of potassium has been fairly tried and has failed, in operating, provided that the tumour can be localised, and that its position does not preclude successful operative interference, even although the diagnosis is not quite clear on the other points, *i.e.*, as regards its exact pathological nature and extent.

Recent surgical experiences have shown that the operation of trephining in man is *per se* attended with comparatively little risk to life; while experimenters have shown that in the lower animals enormous portions of the brain may be removed without causing death. Provided that rigid antiseptic precautions are adopted,[1] there is reason to suppose that in man the risks of removing considerable portions of the brain are very much less than was at one time thought to be the case.

Further — and this point is much more important than any

[1] A comparison of the results obtained by Ferrier in the monkey, without and with strict antiseptic precautions, affords to my mind one of the most striking proofs which has ever been advanced of the immense value of the Listerian method.

number of theoretical considerations — Victor Horsley has demonstrated that some cases of intracranial tumour in man *can* be satisfactorily dealt with in this manner.

The operation of trephining may perhaps, in some cases—but only when the symptoms are urgent and desperate, and when other means have failed—be advisable as a palliative measure. In one case of this kind, in which the diagnosis was attended with great difficulty, but in which, so far as I could judge (and I was confirmed in this opinion by Dr. Hughlings Jackson, who had also seen the patient), the symptoms were due to an organic cerebral lesion, probably a tumour, the operation, which was performed by Dr. Caird, has been so far attended with marked relief. I repeat that trephining, as a palliative measure, is only in my opinion justifiable when the symptoms are desperate, and when other means have failed to give relief.

Palliative Measures.—For the relief of pain, morphia—particularly in the form of subcutaneous injections,—Indian hemp, and croton chloral, free watery purgation,[1] the administration of remedies, such as iodide of potassium and the nitrites, which reduce blood-pressure, and the application of cold in the form of ice-bags[2] to the head—are, in my experience, the most useful. Ergot of rye, which has been recommended by some writers for the relief of headache, has not in my hands yielded any beneficial results.

For the relief of spasms and epileptiform convulsions, bromide of potassium and chloral hydrate are the chief remedies. Localised spasms or attacks of Jacksonian epilepsy, due to syphilitic deposits on the surface of the motor area, are best treated by large doses of iodide of potassium; when very severe, chloral hydrate, or bromide of potassium and chloral hydrate, may be given, with the object of restraining the epileptic seizures until the iodide has time to exert its specific effects.

Apoplectic attacks due to hæmorrhagic extravasation are to be treated in the same way as ordinary apoplexies.

[1] The marked benefit which in some cases follows free purgation is well exemplified in a case which I have reported in the *Edinburgh Medical Journal* for February 1887, p. 688.

[2] In a case of cerebellar tumour, which I have recorded in the *Edinburgh Medical Journal* for June 1879, p. 1073, the patient, a girl aged nine, found out for herself that the application of cold to the head relieved the headache. Her mother stated that the patient used to stand for half an hour at a time with her head under the cold tap.

In pseudo-apoplectic attacks, the application of cold to the head, blistering, free purgation, or venesection, are the best remedial measures.

The paralyses which are caused by intracranial tumours must be treated in the same manner as the paralyses due to other lesions. Epileptiform paralysis rapidly disappears, provided that the localised convulsive seizures with which it is associated can be relieved by appropriate treatment. As already stated, iodide of potassium is the remedy which is most likely in such cases to afford permanent relief.

Paralysis due to a syphilitic lesion of a nerve trunk should be treated, in its earlier stages, by large doses of iodide of potassium alone, or in combination with mercury. In the later stages of cases of this description, and after the system has been thoroughly saturated with the iodide, I have found strychnine, arsenic, and electricity beneficial.

In all cases of intracranial tumour in which double optic neuritis is developed, the administration of iodide of potassium is advisable. Under this treatment the inflammation of the optic papilla is, in some instances, reduced or altogether relieved, and the occurrence of post-neuritic atrophy and blindness thereby prevented. Even when the optic neuritis is so intense as to produce complete blindness, the most marked improvement may take place under this treatment. Dr. Hughlings Jackson, for example, states that "it is well known that optic neuritis may pass off under treatment, leaving good sight, whether there has been a defect of sight or not,—even when there has been complete blindness."

Such marked improvement is probably only to be hoped for in syphilitic cases; for other forms of tumour, with such intense optic neuritis, are usually fatal. On this point I am disposed to agree with Dr. Buzzard, who states "that he did not think that he had ever seen atrophy of the disc ensue in any case of optic neuritis accompanying *syphilitic* disease of the brain in which specific treatment had been adopted before there was any failure of sight. On the other hand, he did not feel thoroughly satisfied that he had often seen successful treatment of swollen discs by iodide or mercury in a case where a syphilitic element was not probably present."[1]

[1] *Transactions of the Ophthalmological Society of the United Kingdom*, vol. i., p. 99.

But it is of the greatest importance to recognise the facts that the most marked improvement may occur even when the papillitis is intense; and that, with the object of preventing post-neuritic atrophy and blindness, iodide of potassium should be freely administered as soon as the inflammatory changes in the fundus are recognised.

It is unnecessary to refer to the treatment of bed sores, cystitis, and other complications and associated lesions.

The external localisation, or the method which is to be adopted in order to ascertain the exact point on the surface of the scalp which corresponds to any given portion of the cortex of the brain underneath, together with the surgical details which it is advisable to adopt in performing the operation for the removal of brain tumours, are described in the following chapter, which Mr. Hare, whose work in this department is well known, has been so kind as to write for me.

CHAPTER XI.

THE SURGICAL TREATMENT OF INTRACRANIAL TUMOURS.

By Arthur W. Hare, F.R.C.S.Ed.

I. There are certain regions within the cavity of the skull that have been recently brought within the bounds of legitimate surgical interference for the presence of neoplasms and inflammatory accumulations. Tumours of the meninges, for the most part originating in cicatricial tissue following an injury, and tumours of the cortical substance itself, are now successfully removed by the surgeon; and circumscribed areas of suppuration in each of these positions, and in the interior of the brain, have been repeatedly operated on with success. As yet these must constitute the limits of legitimate operative interference, if the statistics of recorded cases are to be relied on; for the attempts that have been made to add diffuse suppuration, deep-seated tumours, and localised idiopathic hæmorrhages to the category of operable affections have not been attended with encouraging results. Of the deep-seated tumours, those of the cerebellum seem least certainly included in this prohibition, for the objection in their case is the technical difficulty of the operative procedure, rather than the physiological contra-indications that apply in the case of deep-seated growths of the cerebrum.

II. Having defined in this way the scope of operative surgery, as at present practised, in the region of the cranium for idiopathic lesions, it is important to consider the chief structural features of that region to which the surgeon must have regard in planning his remedial operations.

In the preceding chapters the study of localised function in the brain, and the light thrown thereby on the delimitation of localised lesions, have been fully referred to; and it is now important to inquire in how far this field of knowledge may be available for the purposes of defining the exact situations in which surgical interference

must be undertaken. For this purpose it is necessary to define, within the limits of approximate accuracy, the anatomical relation of various areas of the cerebral cortex to constant external landmarks on the surface of the head. Some satisfactory and practically useful method of defining these important relations must be discovered, if possible, which shall not involve a preliminary reflection of the scalp, before it can be applied, since the appropriate treatment of the scalp itself, and its careful protection during an operation, are of the greatest consequence in securing success in every case of surgical interference with the head. A system solely founded on the recognition of surface markings on the bones of the head will therefore prove unsatisfactory, where exact precision is so all-important. It is true that it may render confirmatory information to the surgeon during the course of his operation, but the primary data on which he must rely are such as he can determine with all the cranial coverings still intact. For this reason the cranial sutures must be left out of count, since they are not accurately discernible in the living subject, and a practical method must be established on the topographical relations of bony eminences which are always available for the purpose, and give sufficient accurate information on the point at issue. Before selecting those landmarks which are of actual value for this purpose, it is necessary to exclude all such bony prominences as have no constant and essential relation with one or other region of the cranial cavity. Further, those selected must bear an uniform relation to the brain-case as a whole, irrespective of varying degrees of relative individual development in the bones, of which they form a part. That bony prominences of the facial region must be absolutely excluded from the calculation, on account of their unequal development in relation to the cranium in different races and in different individuals of the same race, is sufficiently shown by the classic researches of Camper on the varieties of the facial angle as affecting physiognomy. If it be somewhat less obvious, it is equally important to notice that bony landmarks of the lower segments of the skull itself must be for a like reason discarded, since they give no accurate basis for a system of mapping out the surface of the head in relation to the underlying regions of the cortex. If, for instance, the external auditory meatus or the mastoid process be employed for such a purpose, the lines

traced from them upwards over the vault will give inconstant results, according as the frontal zone and the anterior fossa, or the occipital zone and the posterior fossa, are severally more or less developed at the expense of the parietal zone and the middle fossa, to which alone such measurements can accurately apply. The comparison of a well-marked dolicocephalic cranium with one of a brachycephalic type, in regard to the relative development of these three zones or segments, shows clearly to how great an extent such a system is calculated to mislead the surgeon. If these outstanding landmarks be left out of account for the reasons just stated, it will be found that enough still remain for the construction of a clear and sufficiently precise method of localisation. Four bony prominences of the cranium proper are particularly of value. These are—(1.) The glabella, or root of the nose, which bears a definite relation to the anterior limit of the cranial cavity. (2.) The occipital protuberance or inion, which bears a similar relation to the posterior extremity of the cerebral receptacle, and to the upper limit of that containing the cerebellum, since it marks the point of junction of the falx with the tentorium. Between these two poles of the vault, is disposed the whole mass of the cerebrum, and their surface distance apart, on the vertex, is definitely related to the mesial surface measurement of the whole arc of the cerebrum. With an immaterial correction for varying thickness of the skull and scalp, this mesial surface measurement from glabella to inion bears a constant relation to the corresponding cerebral measurement, unaffected by the varied relative expansion of the individual bones of the vault, that is found to exist in all human beings. (3.) The external angular process of the frontal bone is the third bony point that affords reliable evidence on the principle already defined. It bears a definite relation to the antero-lateral extremity of the frontal lobe, and to the point of origin of the fissure of Sylvius; since it marks the plane of the floor of the anterior fossa, the posterior limit of which corresponds to the interval between the frontal and temporo-sphenoidal lobes. (4.) Lastly, the parietal eminence is of similar value, since it marks the point of greatest lateral expansion of the substance of the hemisphere. Though its distance from the vertex varies greatly in different heads, and its relation to other bony points, and even to the bone of which it is a part, are not

uniform, it is still, as has been shown by Turner, a useful landmark. Its cerebral relations are much more constant than its cranial relations, and it is on that account of special value for the purposes of localising the position of cortical areas.

III. With these anatomical data to work upon, the first object is to define the position of the primary fissures of the brain in relation to the surface of the head. The fissure of Rolando claims the first place, inasmuch as those areas of functional representation associated with motor phenomena are grouped around it, and are the most accurately defined of all the cortical areas concerned in localised function. To define the upper end of the fissure, where it joins the great longitudinal fissure, the surface measurement of the scalp should be taken in the middle line from the glabella to the external occipital protuberance. In ordinary adult heads this will vary from eleven to thirteen inches. Measured from before backwards along this line, the distance from the glabella to the top of the fissure is 55·7 per cent. of the length of the whole line. The following sliding scale shows the distance from the glabella to the top of the fissure in heads of ordinary size:—

Distance from glabella to occipital protuberance.	Distance from glabella to top of fissure of Rolando.
In a head of 11 inches,	$6\frac{1}{10}$ inches.
$11\frac{1}{2}$ „	$6\frac{2}{5}$ „
12 „	$6\frac{3}{5}$ „
$12\frac{1}{2}$ „	7 „
13 „	$7\frac{1}{5}$ „

This scale is based on a series of observations made on a number of perfectly fresh trunkless heads, treated by a special method intended to conserve the proper anatomical relation of parts, while giving evidence on this important cranio-cerebral relation. Eleven heads of greatly varying type and proportion were employed in elaborating this uniform scale, and as it was found reliable in cases of extreme variation, it will apply even more accurately to heads of ordinary shape. It depends for its accuracy upon the fact that the pre-Rolandic and post-Rolandic regions of the brain bear in general a uniform proportion to one another as regards extent, irrespective of the size of the brain as a whole. The pre-Rolandic arc is thus a definite

fraction of the whole arc of the brain surface, measured in the mesial plane; and that fraction has been defined by the measurements referred to as ·557 of the whole arc. The whole cerebral arc is represented on the surface of the scalp by the whole line from the glabella to the inion, and the pre-Rolandic arc is similarly represented on the surface by the anterior ·557 of that line. Thus an exact scale based on this fractional relationship can be established applicable to any head. Professor Thane, of University College, London, advocates a modification of this method. He halves the distance from the glabella to the inion, and, having thus defined the middle of the vertex, takes a point half an inch behind it as the top of the fissure. This is not so accurate as a sliding scale, but is sufficiently so for practical purposes in ordinary heads; and, as such, it is to be recommended as the most ready and convenient method in the majority of cases; while for unusually small or large heads a fresh calculation should be made on the data above given.

The localisation of the upper end of the fissure in the middle line of the scalp is by no means sufficient for practical purposes; for it is no less essential to be able to map out its extent and direction. The average measurement *on the scalp* corresponding to the distance between its extremities (leaving out of account its two curves) is $3\frac{3}{8}$ inches in medium-sized heads, and its direction is from above downwards and forwards. The long axis of the fissure forms, with the mesial line, an angle of 67°, the angle opening forwards. To give practical value to the foregoing facts, an instrument has been devised, at the suggestion of Professor Chiene, by Dr. Claude Wilson of Tunbridge Wells. It combines with the scale of measurements for localising the fissure those data representing its length and direction (see Fig. 114). The mode of its application to a patient's head is shown in Fig. 115. In coincidence with Professor Chiene's object in suggesting its construction, the instrument can be applied to a patient in the recumbent posture, as is often essential in cases where operation is necessary.

This instrument is extremely simple in its structure, consisting merely of three strips of flexible metal, and a tape for securing them in position on the patient's head. In its application the broadest transverse strip is applied circumferentially around the head, em-

bracing the glabella and the external angular process in the frontal region, and being firmly secured at the occipital protuberance by means of the tape attached to the eyelet of the strip. A narrower longitudinal strip of metal attached to the first strip at a right angle

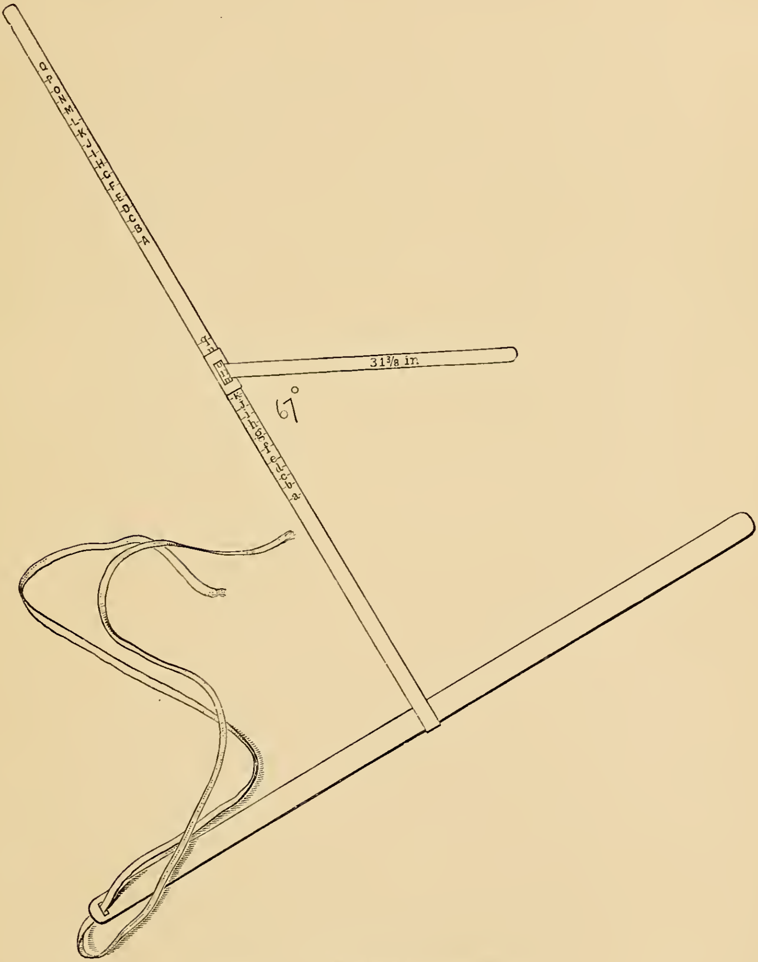

FIG. 114.—Wilson's Cyrtometer.

passes backwards in the middle line over the scalp to the occipital protuberance. This strip is marked with two series of letters: capitals near its occipital end, small letters about the middle of the strip. These two series of letters are related to one another, each to

each, as ·557 is to unity, measuring from the frontal end of the strip. Hence, when any given capital letter on the strip falls upon the prominence of the inion, the corresponding small letter points out the upper limit of the fissure of Rolando in that particular head. A third narrow reversible slip glides forwards or backwards on the

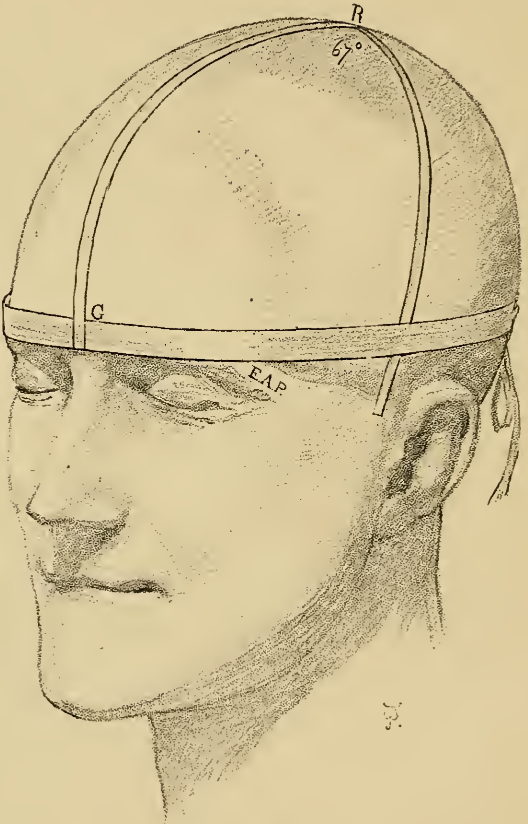

FIG. 115.—Wilson's Cyrtometer *in situ*. *G*, glabella ; *E A P*, external angular process ; *R*, fissure of Rolando—its position and direction marked by the lateral strip of metal.

longitudinal strip, and marks an angle of 67°, opening forwards, thus giving the axis of the fissure. Thus its direction and length can be ascertained on any head as above defined.

The fissure of Sylvius has definite relations both to external landmarks of the head and to markings on the surface of the skull, which

enable the surgeon to localise its position prior to operative interference, and to confirm his view of its locality during the early stages of the operation. To define its position, a line is drawn from the external angular process of the frontal bone backwards to the occipital protuberance, taking *the nearest route* between those two prominences. Such a line droops a little towards the external auditory meatus, in avoiding the greater convexity of the side of the skull that lies in the direct *horizontal* route between the two bony eminences (*EAP–OPr*, Fig. 116). It usually passes over the temporal bone half an inch above the meatus, at which place it corresponds to the floor of the middle fossa of the base of the skull.[1] In front of the meatus it is above the floor of the middle fossa; and behind, it runs parallel to, and usually coincident with, the attachment of the tentorium and the posterior half of the lateral sinus. A measurement of $1\frac{1}{8}$ inch backwards from the angular process along this important line gives the point of origin of the fissure of Sylvius. From this point a straight line drawn to the centre of the parietal eminence marks accurately the course of the posterior limb of the fissure. On reflecting the skin and the temporal fascia and muscle, the surgeon will find the pterion (Fig. 116), or junction of the sphenoid with the frontal, parietal, and temporal bones. From its point of origin under the great wing of the sphenoid, the main line of the Sylvian fissure follows that of the squamo-parietal suture to its highest point, from which the fissure diverges upwards, continuing its course to the parietal eminence. The ascending limb of the fissure, or precentral sulcus, corresponds closely with the upper part of the sphenoido-squamosal suture, the line of which it continues upwards for half an inch, giving off the inferior frontal sulcus at right angles near its upper extremity. An important anatomical detail of this region, which the surgeon does well to keep in mind, is that the middle meningeal artery, after grooving the under surface of the great wing of the sphenoid, passes on to the anterior inferior angle of the parietal, and is distributed to the dura

[1] This point is of special importance to the surgeon, since it has been pointed out by Dr. M'Bride and Mr. Miller that this is the proximal route to adopt in the evacuation of intracranial abscesses originating in the tympanum. The roof of the tympanum, which is usually perforated in these conditions, is here most easily reached, and the pus thus intercepted and evacuated at its source of origin.

mater lining the anterior and superior half of that bone (Fig. 116). If he desire to expose the tip of the temporo-sphenoidal lobe, he should trephine below and behind the line of the artery; if to cut down on Broca's convolution, immediately in front of the same line.

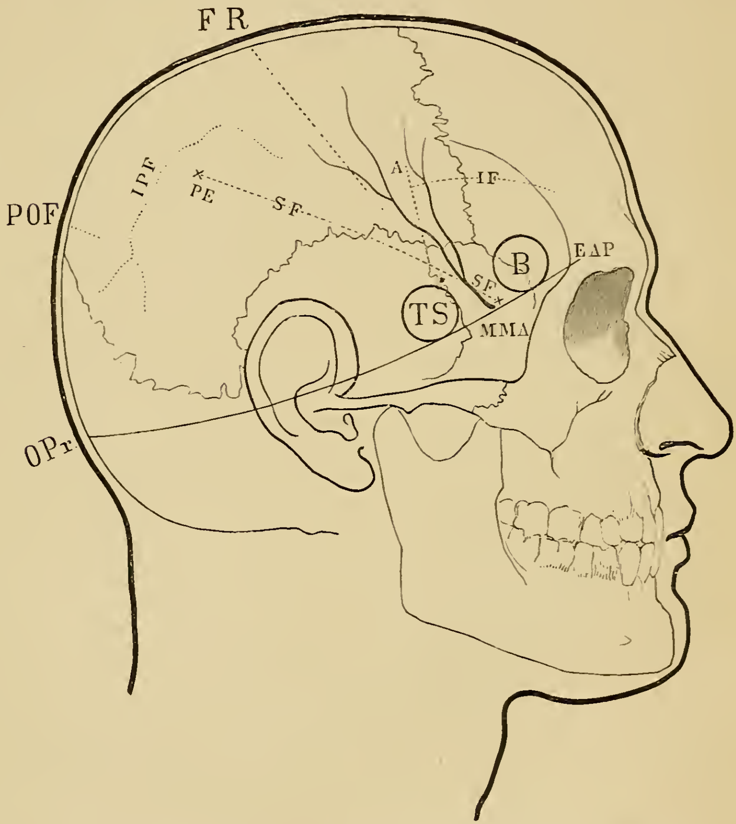

Fig. 116.—Head, skull, and cerebral fissures (adapted from Marshall). *O Pr*, occipital protuberance; *E A P*, external angular process; *S F*, Sylvian fissure; *A*, its ascending limb; *F R*, fissure of Rolando; *P E*, parietal eminence; *M M A*, middle meningeal artery; *T S*, tip of temporo-sphenoidal lobe; *B*, Broca's convolution; *I F*, inferior frontal sulcus; *P O F*, parieto-occipital fissure; *I P F*, intra-parietal sulcus. The pterion region is that where three sutures meet;—those bounding the great wing of the sphenoid where it joins the frontal parietal and temporal bones.

To define the position of the remaining sulci of the brain, the two primary fissures must be mapped out on the scalp as already described. Then the parieto-occipital fissure will be at a point two inches behind the upper end of the Rolandic fissure; and when the

scalp is reflected, that point will be found to be placed a quarter to half an inch in front of the apex of the lambdoidal suture. The fissure is about one inch in length, running at right angles with the the great longitudinal fissure with which its upper extremity is continuous.

The intra-parietal sulcus commences one inch posterior to, and at the level of, the middle third of the fissure of Rolando, having the ascending parietal convolution between it and that fissure. It runs upwards and backwards for about an inch, and then turns directly backwards, forming the lower boundary of the superior parietal lobule. Then, passing below the outer extremity of the parieto-occipital fissure, it enters the occipital lobe, in which it terminates.

IV. In describing the operative procedure for the removal of intra-cranial neoplasms, it is well to follow the rules laid down by Professor Victor Horsley, the able exponent and brilliant exemplar of successful procedure in this department of operative surgery. Rigid asepticity is essential, and is secured by a searching preliminary purification of the scalp, and the employment of complete antiseptic measures and the carbolic spray during the operation. The scalp wound is crescentic in shape, and of large dimensions, the soft parts being turned freely back from the area of the operation. Trephines of large size are used, two contiguous circles of bone being removed, and the isthmus of bone between them sawn through, so as to make one large aperture in the skull. The fragments of bone are carefully preserved in warm antiseptic cloths, and are chipped up and replaced at the end of the operation according to Macewen's method. A white shimmering through the dura and a lack of pulsation indicate that the position of the tumour has been correctly diagnosed. The dura is incised, and the tumour removed with a sharp scalpel if firm, or with a spoon if soft or friable, the hæmorrhage being reduced by the preliminary exhibition of morphia to reduce cerebral congestion, and controlled locally by the pressure of sponges. When it has ceased, the dura is stitched with catgut, the fragments of the cranial bones returned, and the scalp very accurately stitched. No drain is placed in the wound, which is carefully dressed and accurately bandaged, so as to secure steady and equable pressure. Primary union is looked for; and should it not occur, or should hernia cerebri ensue, a failure in the

aseptic precautions is betokened. Should there be tension, a strictly purified probe is introduced at a remote angle of the scalp wound, and the escape of a little serum permitted. This is usually sufficient to ensure satisfactory healing, in Professor Horsley's experience.

If the operation be for abscess, the cause is not infrequently septic bone disease. In this case, after evacuating the contents, efficient drainage must be maintained by a rubber drainage tube, and the abscess cavity washed out daily with a weak antiseptic lotion, till contraction and healing ensue, or till the discharge becomes odourless, when the daily dressing and irrigation may be omitted.

In all cases of operation on the cranium, the general treatment requires no less attention than the local; and, if possible, all patients should be subjected to preparatory treatment and observation before an operation is undertaken.

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